EEG AND NEUROIMAGING IN DEVELOPMENTALLY NORMAL CHILDREN WITH AFEBRILE SEIZURES

Dissertation Submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

In fulfillment of the regulations for the award of the degree

M.D.(PEDIATRICS)

DEPARTMENT OF PEDIATRICS

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

COIMBATORE, TAMILNADU

APRIL 2016

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In fulfillment of the regulations for the award of the degree

M.D. PEDIATRICS

GUIDE

DR.K.JOTHILAKSHMI

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PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

APRIL 2016

DECLARATION

I hereby declare that this dissertation entitled **"EEG AND NEUROIMAGING IN DEVELOPMENTALLY NORMAL CHILDREN WITH AFEBRILE SEIZURES"** was prepared by me under the guidance and supervision of **Dr.K.JOTHILAKSHMI**, Professor of Pediatrics, PSGIMS&R, Coimbatore.

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, Chennai in fulfillment of the university regulations for the award of MD degree in Pediatrics. This dissertation has not been submitted elsewhere for the award of any other Degree or Diploma.

Dr.S.VIGNESH

CERTIFICATE

This is to certify that the thesis entitled **"EEG AND NEUROIMAGING IN DEVELOPMENTALLY NORMAL CHILDREN WITH AFEBRILE SEIZURES"** is the bonafide work of **Dr. S.VIGNESH**, done under my guidance and supervision in the Department of Pediatrics, PSG IMS&R, Coimbatore in fulfillment of the regulations laid down by The Tamilnadu Dr. M.G.R. Medical University for the award of MD degree in Pediatrics.

Dr. K. JOTHILAKSHMI

Professor

Department of Pediatrics

PSGIMS&R

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July 24, 2014

To
Dr S Vignesh
Postgraduate
Department of Paediatrics
PSG IMS & R
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The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 18th July, 2014 in its expedited review meeting held at IHEC Secretariat, PSG IMS&R, between 10.00 am and 11.00 am, and discussed your study proposal entitled:

"Neuroimaging and EEG in developmentally normal children with afebrile seizures"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Assent form
4. Parental consent form
5. Confidentiality statement
6. Application for waiver of consent
7. Data collection tool
8. Permission letter from Head of the Institution
9. CV
10. Budget

After due consideration, the Committee has decided to approve the study.

The members who attended the meetings at which your study proposal was discussed are as follows:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member - Secretary	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member –Social Scientist	Male	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes

The approval is valid for one year.

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

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Yours truly,

24.7.14

Dr S Bhuvaneshwari
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INTRODUCTION

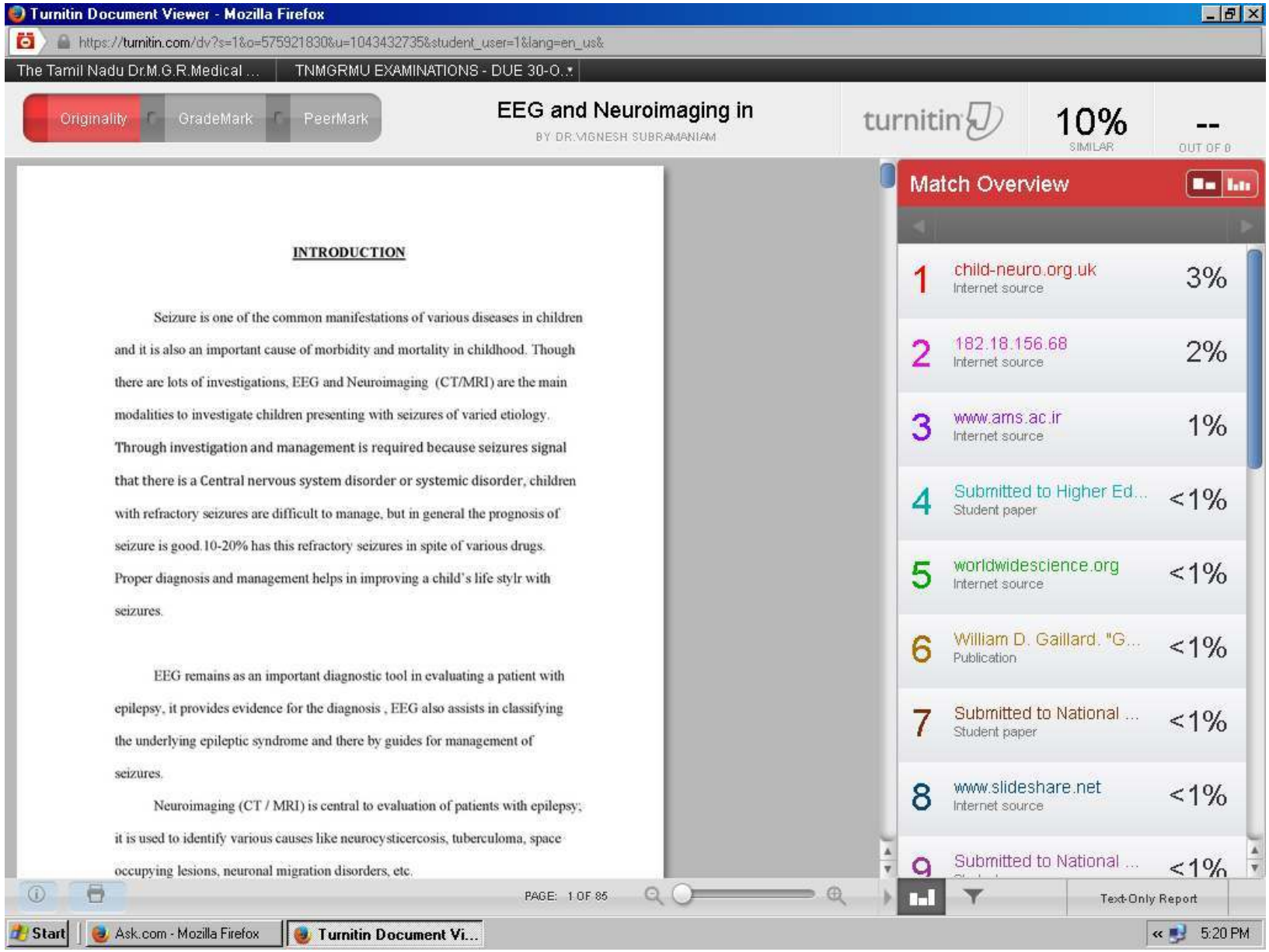
Seizure is one of the common manifestations of various diseases in children and it is also an important cause of morbidity and mortality in childhood. Though there are lots of investigations, EEG and Neuroimaging (CT/MRI) are the main modalities to investigate children presenting with seizures of varied etiology. Through investigation and management is required because seizures signal that there is a Central nervous system disorder or systemic disorder, children with refractory seizures are difficult to manage, but in general the prognosis of seizure is good (10-20% has this refractory seizures in spite of various drugs. Proper diagnosis and management helps in improving [illegible] with seizures.

EEG remains as an important diagnostic tool in evaluating a patient with epilepsy, it provides evidence for the diagnosis , EEG also assists in classifying the underlying epileptic syndrome and thereby in guides for management of seizures.

Neuroimaging (CT / MRI) is essential in evaluation of patients with epilepsy, it is used to identify various causes like neurocutaneous, leukodystrophies, space occupying lesions, neuronal migration disorders, etc.

There are few current guidelines for neuroimaging in children,

1. Focal onset of seizure at any age either in history or EEG
2. Generalized seizures in first year of life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INTRODUCTION
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EEG and Neuroimaging in Developmentally Normal Children With Afebrile seizures

ABSTRACT

Introduction: Seizure is one of the common manifestations of various diseases in children and it is also an important cause of morbidity and mortality in childhood. Though there are lots of investigations, EEG and Neuroimaging (CT/MRI) are the main modalities to investigate children presenting with seizures of varied etiology.Proper diagnosis and management helps in improving the every day life of children with seizures.

Objectives: To correlate the EEG and Neuroimaging findings in developmentally normal child with afebrile seizures.

Study design: A part retrospective and part prospective observational study

Material and Methods: A total of 128 children (1 to 5 years of age), who presented with afebrile seizures were included, they underwent EEG and Neuroimaging (CT or MRI) as decided by the treating physician, records analyzed for retrospective cases.

Results: Out of 128 children 70 were boys and 58 were girls, children between 1-5 years of age were more. Generalized seizure was noted in 64.8% of children and focal seizures were noted in 35.2% of children. EEG abnormality was seen in 108(84.4%) of children, Neuroimaging abnormality in 29(22.7%) children, Most common EEG abnormality in Bilateral generalized epileptiform

activity (43.7%), followed by Sharp spike waves and Sharp waves. and Gliosis was the common neuroimaging abnormality, on correlating Neuroimaging and EEG, 9 out 20 children with normal EEG (45%) had abnormal neuroimaging (P value=0.009). 84.3% of children were on single anticonvulsant in spite of both EEG and Neuroimaging abnormalities, Valproate was the commonly used anticonvulsant.Family history was more in common in children with focal seizures (26.7%).

Conclusions: 1. Among 128 children in the study 108(84.4%) had an abnormal EEG, 29 (22.7%) out of 128 had abnormal neuroimaging. 2. The most common EEG abnormality was Bilateral Generalized epileptiform activity, which was seen in 43.7% of children, and Gliosis was most common neuroimaging abnormality, which was seen in 27% of children. 3. The incidence of getting abnormal neuroimaging is similar in both focal and generalized seizures. 4. For seizure control 82.5% of children with abnormal EEG required only one Antiepileptic drug.

Keywords: Seizure, Neuroimaging, EEG, Afebrile seizures.

INTRODUCTION

Seizure is one of the common manifestations of various diseases in children and it is also an important cause of morbidity and mortality in childhood. Though there are lots of investigations, EEG and Neuroimaging (CT/MRI) are the main modalities to investigate children presenting with seizures of varied etiology. Through investigation and management is required because seizures signal that there is a Central nervous system disorder or systemic disorder, children with refractory seizures are difficult to manage, but in general the prognosis of seizure is good.10-20% has this refractory seizures in spite of various drugs. Proper diagnosis and management helps in improving the every day life of children with seizures.

EEG remains as an important diagnostic tool in evaluating a patient with epilepsy, it provides evidence for the diagnosis, EEG also assists in classifying the underlying epileptic syndrome and there by guides for management of seizures.

Neuroimaging (CT / MRI) is central to evaluation of patients with epilepsy; it is used to identify various causes like neurocysticercosis, tuberculoma, space occupying lesions, neuronal migration disorders, etc.

These are few current guidelines for neuroimaging in children,[55]

1. Focal onset of seizure at any age either in history or EEG
2. Generalized seizure in first year of life.
3. Focal or neurological deficits.
4. Loss of seizure control with current anti epileptic drug.
5. Change in seizure pattern, which may suggest some underlying lesion.

There are numerousstudies, which have been done correlating EEG and Neuroimaging in children presenting with seizures, with conflicting results. However there are very few studies from India on this subject. This study was done to determine the correlation of EEG and Neuroimaging in developmentally normal children with afebrile seizures.

AIM

AIM:

To study Neuroimaging and Electroencephalogram findings in developmentally normal children presenting with afebrile seizures.

OBJECTIVE:

To correlate the EEG and Neuroimaging findings in developmentally normal child with afebrile seizures.

REVIEW OF LITERATURE

DEFINITIONS:

SEIZURE: [1,2]

A Seizure or convulsion is a paroxysmal, time-limited change in motor activity and/or behavior that results from abnormal electrical activity in the brain.

EPILEPSY:

Epilepsy is a chronic disorder in brain. A seizure is a transient sign, which occurs due to abnormal or increased neuronal activity in the brain. Epilepsy is considered when an individual has any one of the following.[1,8]

1. At least 2 unprovoked seizures, which occurs more than 24 hours apart.
2. Children with 1 unprovoked seizure and with increased risk of getting similar episodes of unprovoked seizure.

PREVALENCE: [37]

Seizures occur in 10% of children. The lifetime incidence of getting epilepsy is around 3% and more number of cases begins in their childhood. Most of the Children outgrow epilepsy because the annual prevalence rate is 0.5 to 0.8%. However and a recent meta-analysis suggests that the prevalence rate is 5.59 per 1000 population, this is similar to rate in developed countries.

INCIDENCE OF EPILEPSY:[25]

There are very few global studies to establish total population of age adjusted incidence of epilepsy- 24 to 53/ 1,00000 person years. Total population studies about the first diagnosis of single unprovoked seizures among children ranges from 26 to 70/ 100000-person years. Several studies from the past 2 decades (1986-2005) denotes than incidence of unprovoked seizures are generally consistent in the developed countries and incidence among the developing countries are comparatively higher- 114/100000 person years.

This has mostly been recorded in the rural areas. They show 2 to 3 times higher incidence than the developed countries. The overall incidence of seizures including the febrile seizures and absent seizures were recorded to be higher than other reports- 190/100000 person years, still neurologically confirmed cases among them are 30% less than the overall incidence.

In a study conducted in France[52], more than half of afebrile seizures do not meet the criteria of epilepsy. When comparing the cause of acute asymptomatic seizures, studies are very less. In developing countries the incidence is high compared with the industrialized countries.

ETIOLOGY: [8]

Seizures are mainly due to the damage in he cerebral cortex; even a small damage can cause seizure. It is self-limiting it will cease soon after the inciting process stops.

It can occur at any area in the brain but mostly it arises from the neocortical grey matter and the limbic system. Some of the seizures can also arise from the sub cortical regions. In the limbic system is mainly from hippocampus and amygdala specifically the hippocampus and amygdala. The thalami, basal ganglia, and posterior fossa structures, including the cerebellum, are considered incapable of generating seizures but participate as neuromodulatory influences and act as a relay station from a cortical or limbic origin. Most of the EEG cannot detect the seizure, which arises from the subcortical region. This situation is similar to neonatal seizure because in that it is thought abnormal activity is mainly from the brain stem and not the electrical activity in the brain.

An epidemiologic study[52,41] from, Minnesota reported that the highest percentage of individuals (children and adults) with epilepsy fell into the idiopathic/cryptogenic category (ie, presumed genetic or unknown etiologies), other causes are infection, neoplasms, degenerative disease of the brain and condition which occurs as mental retardation and CP which usually will be seen from younger age. In children less than 14 year age group, a developmental etiology was more common.

As these data for most of these studies were collected in the past before advent of advanced neuroimaging and molecular genetics; it is likely that the unknown group will be smaller in future studies, as subtle brain malformations, including dysplastic lesions and migrational disorders, are increasingly recognized with help of recent imaging facilities. Advancement in genetic research also will allow us to identify the chromosomal location and the abnormal gene product in many familial type of epilepsies, thereby establishing entirely new classification categories with the emphasis on genetic origins of seizures.

A small but not insignificant group of children have chronic seizures due to acute hypoxic and toxic/metabolic events; infections, particularly bacterial; and vascular lesions. With the increase in child abuse, head trauma is becoming an increasingly common cause of chronic seizures.

INTERNATIONAL CLASSIFICATION OF CHILDHOOD SEIZURES

Previous classification of ILAE:[8]

I. PARTIAL SEIZURES
- Simple partial (consciousness retained)
  - Motor
  - Sensory
  - Autonomic
  - Psychic
- Complex partial (consciousness impaired)
  - Simple partial, followed by impaired consciousness
  - Consciousness impaired at onset
- Partial seizures with secondary generalization

II. GENERALIZED SEIZURES
- Absence seizures
  - Typical
  - Atypical
- Generalized tonic clonic
- Tonic
- Clonic
- Myoclonic
- Atonic
- Infantile spasms

III.UNCLASSIFIED SEIZURES

REVISED CLASSIFICATION OF SEIZURES (ILAE 2010) [1]

1.Focal seizures

- Focal sensory seizures
- Hemiclonic seizures
- Secondary generalized seizures.

2.Generalized seizures

- Clonic seizures
- Tonic seizures.
- Tonic clonic seizures.
- Myoclonic seizures
- Myoclonic atonic seizures.
- Negative myoclonus.
- Atonic seizures.

3.Unknown

FOCAL SEIZURE WITHOUT IMPAIRMENT OF CONSCIOUSNESS:[1]

This was previously known as simple partial seizures. The most common feature of a focal seizure without impairment of consciousness is motor activity. It usually involves the face, neck and extremities; the usual movements present during this episode are tonic or clonic movement. In this focal seizure turning of head and conjugate eye movements are common. This seizure usually persists for only 10 to 20 seconds. During the seizure episode patient usually remains conscious, this is the main distinguishing feature of a focal seizure with out impairment of consciousness from other type of seizures.

FOCAL SEIZURE WITH IMPAIRMENT OF CONSCIOUSNESS: [1]

This was previously known as complex partial seizures.This seizure may begin as focal seizure without impairment of consciousness with or with out aura leading to impairment in conscious level. There will be an predisposing aura which involves fear, epigastric region discomfort or unpleasant feeling. If there is a history of aura it usually indicates focal type of seizures. Most of the children with focal seizures experience the aura.

Spreading of the epileptiform discharge during this type of seizure can result in secondary generalization with a tonic-clonic convulsion. The average duration is 1-2 min, which is considerably longer than a absence seizure.

ABSENCE SEIZURES: [1]

Absence seizure are most common in children more than 5 years of age, usually girls are mostly affected. It involves cessation of motor activity or speech, sometimes just a vacant stare, absence seizure can last more than 30 seconds. It can also present jus with flickering of eyelids. Usually these children will not experience post ictal confusion or aura.

Hyperventilation for 3-4 min routinely produces an absence seizure. The EEG shows a typical 3/sec spike and generalized wave discharge. Complex(atypical) absence seizures have associated motor components consisting of myoclonic movement of the face, fingers, or extremities and,on occasion, loss of body tone. These seizures produce atypical EEG spike and wave discharges at 2-2.5/ sec.

GENERALIZED MOTOR SEIZURES:[1]

These seizures are common and usually predisposed by an focal seizure with secondary generalization. As in of a focal seizure these children can also present with aura, which suggests the focal onset of the seizure. In children with generalized motor seizures history aura should not be missed because it can help in identifying the site of lesion.

Patients suddenly lose consciousness and , in some cases, emit a shrill, piercing cry, Their eyes roll back, their entire body musculature undergoes tonic contractions, and they rapidly become cyanotic in association with apnoea. The clonic phase of the seizure is heralded by rhythmic clonic contractions alternating

with relaxation of all muscle groups. The clonic phase slows towards the end of the seizure, which usually persists for a few minutes, and patients often sigh as the seizure comes to an abrupt stop. During the seizure children may bite their tongue, but rarely vomit. Loss of sphincter control,particularly the bladder, is common during a generalized tonic clonic seizure. The postictal phase is often associated with vomiting and an intense bifrontal headache.

MYOCLONIC EPILEPSIES OF CHILDHOOD:[1]

This disorder is characterized by repetitive seizures consisting of brief, often symmetric muscular contractions with loss of body tone and falling or slumping forward, which has a tendency to cause injuries to the face and mouth. Myoclonic epilepsies include a heterogenous group of conditions with multiple causes and variable outcomes. Atleast five different subgroupings can be identified; these represent a broad spectrum of myoclonic epilepsies in the pediatric population.

TYPES OF MYOCLONIC EPILEPSIES:[1]

- Benign myoclonus of Infancy
- Typical myoclonic epilepsy of early childhood
- Complex myoclonic eplilepsies
- Juvenile myoclonic eplilepsy
- Progressive myoclonic epilepsies

INFANTILE SPASMS:[1]

Infantile spasms are more common in infants between 4 to 8 months of age. They usually have an very short episodes of contractions involving the neck, trunk and the extremities.There are at least three types of infantile spasms;flexor, extensor, and mixed.

Infantile spasm is typically classified into two groups: Cryptogenic and symptomatic. A child with cryptogenic infantile spasms has an uneventful pregnancy and birth history as well as normal developmental mile stones before the onset of seizures. The neurologic examination and the Computed tomography or MRI will usually be normal in these children, and there are no associated risk factors. Approximately 10-20% of the infantile spasms are classified as cryptogenic, and the remainder is classified as symptomatic. Symptomatic infantile spasms are related directly to several prenatal,perinatal and postnatal factors.

LANDAU – KLEFFNER SYNDROME[1]

Landau kleffner syndrome usually common in boys, the mean age of onset is 5 ½ years. This syndrome is usually very rare. In a previously normal child there will be sudden loss of language skills. This syndrome is usually associated with seizure disorder also (70%). The common features of this syndrome are irritability, behavioral problems, these children will have an normal hearing. This can occur as a various type of seizures including focal, generalized, tonic clonic or even as an absence seizure. EEG will show an high amplitude spike wave and it

occurs in both the temporal lobes, CT and MRI scan will be normal. PET scan can reveal the unilateral hypo or hyper metabolism.

GENERAL CLINICAL CHARACTERISTICS:[1,8]

Seizures are usually similar and it can occur randomly at any time of the day, and are rarely precipitated by specific environmental, psychological, or physiological events. Some children have several different types of seizures, but most of them have one type that expresses itself in partial or complete form. for example, a full seizure may be characterized by flashing lights in one visual field (a focal seizure with occipital lobe onset), followed by deviation of the eye (version, caused by spread to the neighboring association cortex), then by loss of awareness of the surroundings associated with automatic behavior (motor automatisms such as lip- smacking and swallowing movements) caused by spread to the temporal lobe limbic system of both hemispheres, and a few seconds later culminates in to a bilateral convulsive seizure. In some occasions, the patient may experience only the first few stages of the seizure.

To understand the location of the seizure or the area involved parents must be asked about the predisposing factor of the seizure, like history of aura or about the first stage of the seizure. But this often difficult or not possible in younger children.

Certain group of children usually experiences the episode of seizures only after some stimulus like high noise, flashing of bright lights, or sometimes-reflex

seizures. Most of the children will have seizure during sleep or soon after getting up from sleep.

CLINICAL BEHAVIOR DURING SEIZURES AND NONEPILEPTIC EVENTS :[1,8]

There are some general rules which help in identifying seizures from other events which usually mimic like seizures, like psychogenic activities, paroxysmal behavior of the children and also cardio vascular dysfunction, these are common condition which can mimic like seizure.

Few examples as follows:[1,8]

- In a normal child cardiogenic causes occurs in child with a sudden loss of tone with or with out impairment of consciousness level. During an atonic seizure child will make a protective move to avoid the falling. However, these seizures usually occur in a neurologically abnormal child (eg, Lennox-Gastaut syndrome, other developmental encephalopathies).
- If the child is cyanosed during the episode then it usually due to hypoxia caused by the generalized motor seizures. Children Non epileptic events like syncope will be pale. So any color change during the episode of seizure must be noted. To rule out this history must be clearly obtained from the mother or father of the child.
- If the parents give a history of irritable or consistent crying before the seizure episode breath holding spell must be considered.

- Due to excess vagal tone or an unexpected blow to the head children usually experience a sudden bradycardia with pallor or even a child may collapse, this will usually be very brief episode, this is an benign pediatric syndrome known as Pallid infantile syncope, in this condition children will usually experience a spontaneous recovery.

- True absence seizures occur during an conversation, while eating or when the child is playing. This absence seizure cannot be stopped by tactile stimulation or calling the child with name. Mostly children with ADHD have similar episodes but they can be interrupted. Absence seizures last for 10 – 30 seconds, children with this seizure will experience a multiple episodes in a day. This should be differentiated from pseudoabsence or from daydreaming.

MECHANISM OF SEIZURES:[1,2]

The clear mechanism of seizure is not known. For a child to develop a seizure multiple physiological factors are responsible, for initiation of a seizure certain group of neurons generate a significant burst discharge and a GABAnergic inhibitory system. Excitatory amino like glutamate and aspartate play a significant role in neuronal excitation which acts on a specific cell receptors.

Seizures may arise from areas of neuronal death, and these regions of the brain may promote development of novel hyperexcitable synapses that can cause seizures. For example, lesions in the temporal lobe cause seizures, and when the abnormal tissue is removed surgically, the seizures are likely to cease. Further, convulsions may be produced in experimental animals by the phenomenon of kindling. In this repeated subconvulsive stimulation of the brain (e.g., amygdala) ultimately leads to a generalized convulsion. Kindling plays an major role in development of epilepsy in humans following an injury. It has been understood that recurrent seizure activity ina abnormal temporal lobe causes transmission of stimulus to the other lobe via corpus callosum and causing seizures.

In infants seizures are more common. Certain seizures in the pediatric population are age specific (e.g., infantile spasms); this is mainly because of the underdeveloped brain, because seizures are less common in adults and older children, Genetic factors account for at least 20% of all cases of epilepsy. Using linkage analyses, the chromosomal location of several familial epilepsies has been identified, including benign neonatal convulsions (20q and 8q), juvenile

myoclonic epilepsy (6p), and progressive myoclonic epilepsy (21q22.3). The genetic defect of benign familial neonatal convulsions has been characterized by the identification of submicroscopic deletion of chromosome 20q 13.3. Furthermore, the substantianigra has an integral role in the development of generalized seizures. Electrographic seizure activity spreads within the substantianigra, causing an increase in uptake of 2-deoxyglucose in adult animals, but there is little or no metabolic activity within the substantianigra when immature animals have a convulsion. It has been proposed that the functional immaturity of the substantianigra may have a role in the increased seizure susceptibility of the immature brain. The GABA sensitive substantianigra pars reticulata neurons play a small role in preventing seizures. It is likely that substantianigra outflow tracts modulate and regulate seizure[1].

INVESTIGATIONS:[1]

The purpose of diagnostic evaluation in a child with seizure is to help in diagnosis and provide evidence for the same. In a child with afebrile seizures investigations play a critical role.

Most children with afebrile seizure require no investigations other than EEG and structural brain imaging. Routine lab investigation are not much of important in evaluating the child with afebrile seizures serum electrolytes, metabolic screening of urine toxicology and blood sugar can be individualized and it can be done if the child requires. In children with first episode of unprovoked or afebrile seizures, lumbar puncture doesn't play a major role. Because it mostly helpful in children with suspected meningitis, encephalitis, sepsis or in children with SAH.

The two major modalities required in child with afebrile seizure include

1. **ELECTROENCEPHALOGRAM**
2. **NEUROIMAGING (CT/MRI)**

ROLE OF EEG IN EPILEPSY[33,36,40,51]

EEG was first discovered in the late 1920, now it has developed in to a major tool in diagnosing a child with seizure. Now it is digitalized with video and other investigative modalities.

Electroencephalography is a continuous recording of electrical activity between reference electrodes placed on the scalp[1].An EEG is recommended as part of the neuro diagnostic evaluation of the child with an apparent first

unprovoked seizure[1]. EEG abnormalities help in clinicaldiagnosis of seizure, and also in the diagnosis of specific syndromes and predict seizure recurrence in the patient, but a normal EEG does not rule out epilepsy. The EEG interpretation is reliable only when it is well recorded and interpreted by an experienced person in EEG reading.

In children with unexplained cognitive, neurobehavioral or scholastic deterioration an EEG may help in diagnosis of specific disorders like SSPE, or epileptic encephalopathies like electrical status in slow wave sleep (ESES), and non- convulsive status epilepticus.

EEG is used[1,33,2]

- To help establish the likely diagnosis of epilepsy
- To help establish the type of epilepsy
- To help identify possible precipitants to epileptic seizures
- To investigate the cause of cognitive decline
- To help localize the onset of focal seizure
- To help predict the likelihood of recurrence after an initial seizure
- To monitor treatment including the time of drug withdrawal

HOW AN EEG CAN BE DONE[1,36,40]

EEG should be done 3 to 4 days after anseizue this is to avoid the Post ictal slowing of the waves.

A sleep EEG after deprivation should be part of all routine recordings in children more than 3 years of age.

Activation procedures like hyperventilation and photic stimulation can be done .

Antiepileptics can be taken before an EEG there is no recommendation for with holding it.

Video-EEG is useful in differentiating non-epileptic events from true seizures and for pre-surgical evaluation.

Interictal EEG is usually recorded for 10-60min[2]

EEG tracing is analyzed in terms of background activity and paroxysmal activity[2]

Background rhythms include various physiological rhythms like

1. Delta waves – 1 to 3/ sec (occur in very deep sleep, infancy)
2. Theta waves – 4 to 7 / sec (occur in temporal and parietal regions in children normally)
3. Alpha – 8 to 12 / sec (occur in normal adults when in awake with eyes closed and quiet, recorded in occipital region)
4. Beta – 13 to 20 / sec (occur during mental activity and tension, recorded from parietal and frontal regions) [1]

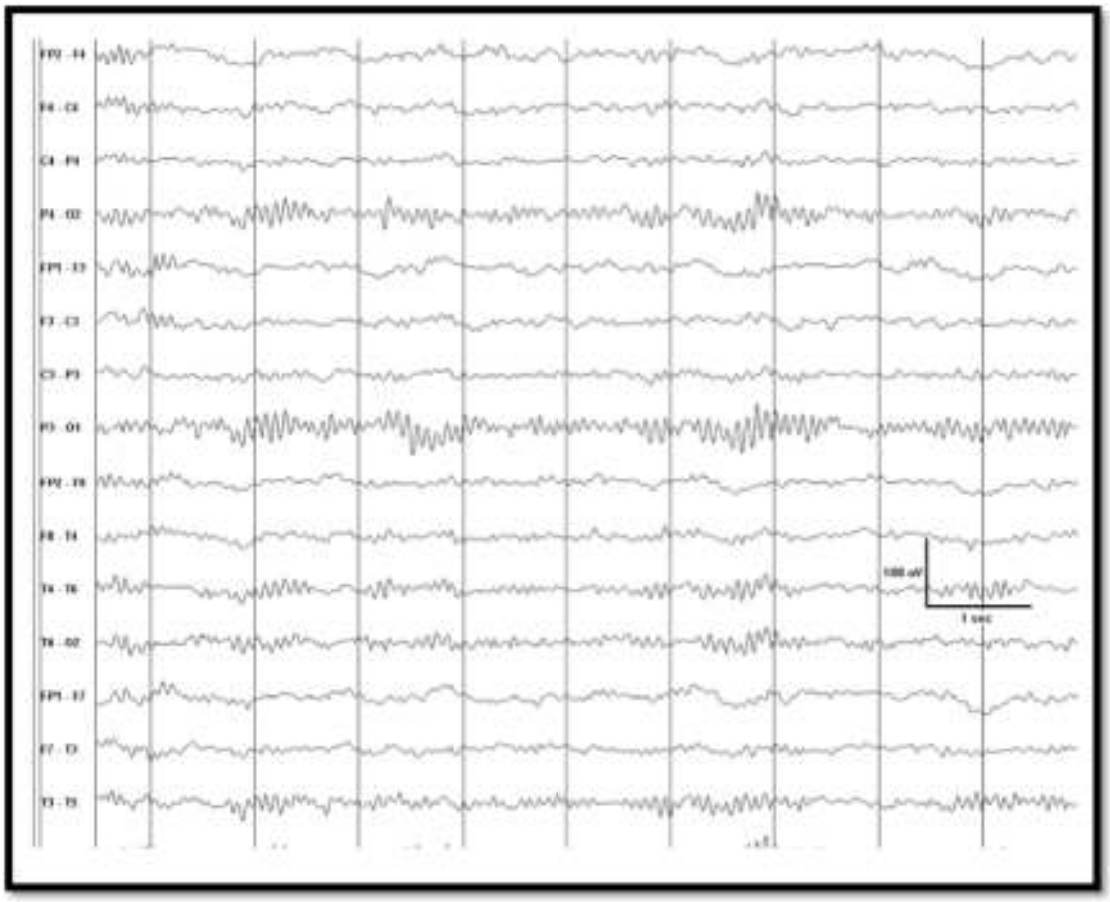

Fig 1 :This is a normal Alpha rhythm seen in a 8 year old boy.

Background rhythms are slower in children and become faster with maturity. As an older child passed from alert to drowsy to light sleep and then to deep sleep, a drop out in the alpha rhythm occurs followed by progressive slowing[2]

High voltage slow and sharp waves (K complexes) and sleep spindles (regular 12 – 14/sec waves) confined to the central regions occur during sleep in normal EEG[2]

Paroxysmal activity stands out from the background. Abnormal paroxysmal activity can be epileptiform or non- epileptiform abnormalities. They are abrupt and short-lived giving rise to spikes and sharp waves. Interictal spikes and waves are often followed by slow waves constituting spikes and wave complexes[2]

Demonstration of paroxysmal discharges on EEG during a clinical seizure is diagnostic of epilepsy. Interictal EEG is normal in 40% of patients.[1]also 2.2 to 3.5% of normal children have epileptiform abnormalities[2].

Certain EEG abnormalities are activated by maneuvers like sleep following partial sleep deprivation[2] or drug induced sedation, hyperventilation and photic stimulation.

Sleep (especially light sleep) is a powerful activator of many EEG abnormalities and if an awake recording is unhelpful it is usually worth obtaining a sleep recording. This can be achieved using natural sleep, following partial sleep deprivation or by drug induced sedation. When drugs induce sleep it should be remembered that these might influence the EEG beyond causing sleep. Benzodiazepine and barbiturates often cause excessive fast activity and suppress many EEG abnormalities: They are therefore best avoided.

Hyperventilation, which should be included in the protocol for standard EEG recordings, is of particular use in investigating subjects with idiopathic generalized epilepsies in whom it frequently activates generalized spike-wave discharges.

Photic stimulation should also routinely be applied during standard EEG recordings. Photoparoxysmal responses are epileptiform abnormalities in which spike and/or spike-wave discharges are produced by intermittent photic stimulation. They are usually generalized but can be confined to the occipital regions. Generalized photoconvulsive responds are most often seen in subjects with idiopathic generalized epilepsies but occur in a variety of other epilepsies and rarely in normal children. Occipital photoconvulsive responses are occasionally seen in subjects with occipital lobe seizures.

EEG FINDINGS IN SPECIFIC CONDITIONS: [33,36]

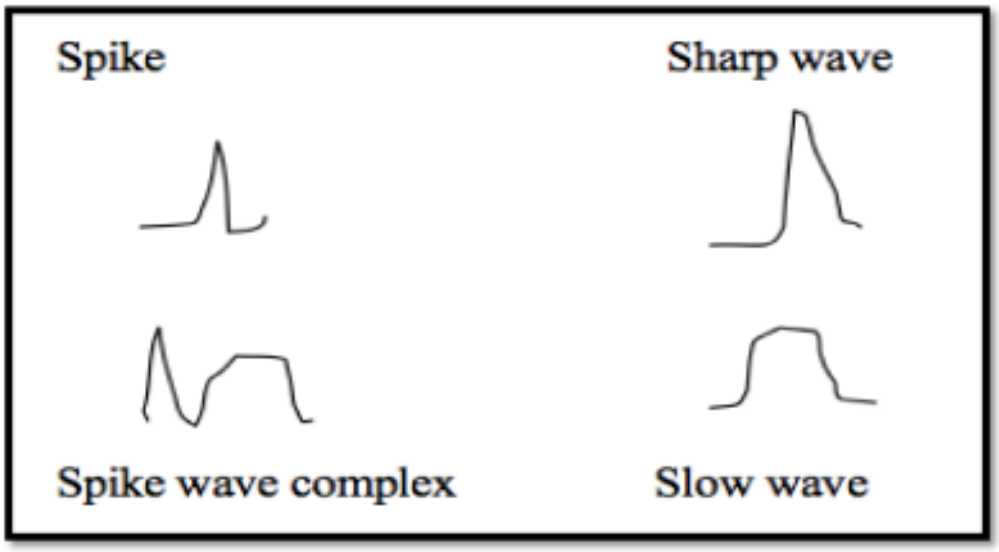

Fig 2: different wave pattern in EEG

Generalized seizure – Usually has generalized epileptiform discharges.

Focal seizures – Usually has focal epileptiform discharges.

Epileptic encephalopathies – Generalized background abnormalities.

Myoclonic epilepsy – Bursts of poly spikes.

Absence seizure – 3 per second spike and wave discharge precipitated by hyperventilation.

Hypsarrhythmia – High voltage generalized chaotic slow waves.

Benign epilepsy of childhood – Centro temporal spike.

Subacutesclerosingpanencephalitis – Periodic epileptiform discharges recurring at similar intervals throughout the record.

Herpes encephalitis – Periodic lateralized slow waves or high voltage complexes.

Fig 3: EEG showing sharp waves

Fig4: EEG shows bilateral epileptiform activity.

ICTAL EEG[1]

During routine EEG recording it is rare to record a seizure. Ictal records require prolonged recording for 24 hours or more and even then are only likely to be obtained if seizures are frequent. They are two types – Ambulatory cassette EEG and Video EEG recording.

Prolonged recordings are useful to:[1,10]

- It helps to decide if paroxysmal clinical events are epileptic.
- To localize the onset of focal seizures. This is usually done as a part of presurgical workup in refractory epilepsy and requires video telemetry rather than a cassette recording.
- To establish the frequency of seizures and interictal epileptiform discharges. This may be useful if a child with epilepsy is performing less well at school than expected and it is considered that this might reflect under recognized seizure activity.

Occasionally children with epilepsy show a stagnation or decline in their cognitive performance. The EEG can be useful in investigating the possible role of epileptiform activity causing this. In some children, especially those with idiopathic generalized epilepsies, frequent interictal and subtle ictal discharges are responsible and can be detected on prolonged EEG recordings, preferably with simultaneous video recording. In others, electrical status during slow wave sleep may be responsible for cognitive problems and this possibility should be investigated by a sleep recording. Finally, some children with apparent cognitive decline are in non-convulsive status epilepticus, EEG can detect.

NEUROIMAGING[1,9]

The prevalence of abnormal neuroimaging in an adult with a new-onset seizure is 34% to 45%. However, the role of neuroimaging in children presenting with first afebrile seizure is still not well defined. Based on several studies conducted the prevalence of abnormal neuroimaging in children with a new onset afebrile seizure is estimated to be 0% to 21%

COMPUTED TOMOGRAPHY: [1,9]

Computed tomography (CT) is the main neuroimaging modality, it is done when a child presents with new onset seizure in a emergency. It is cost effective and easily available and very less time consuming; with the help of CT we can evaluate the acute neurological problems if it needs immediate management. Usually CT is done to rule out intracranial bleeds, structural anomalies, space occupying lesions, and calcifications. Innon-emergency situation MRI can be done because it has more yield and sensitivity is high.

MAGNETIC RESONANCE IMAGING:[1,9,53]

Neuroimaging must be done in all patients with epilepsy. MRI helps in finding the structural lesion responsible for the epilepsy, In elective situation MRI is the modality of neuroimaging Most patients suspected of having had an epileptic seizure should have a neuroimaging study.Which not only has higher sensitivity than CT for most epileptogenic lesions but also better spatial resolution and soft tissue contrast.MRI allows imaging in multiple planes as well as functional cerebral assessment through different techniques.

Exceptions to the need for neuroimaging include children with simple febrile seizures and children whose clinical history and EEG are consistent with benign partial epilepsy of childhood or idiopathic generalized epilepsy.

MRI enables the following brain conditions associated with epilepsy to be detected.[2]

1 Brain malformations and maldevelopments

2 Vascular disorders such as arteriovenous malformations

3 Areas of sclerosis and gliosis associated with old infarcts, hypoxic ischemic insults and infection

4 Tumors

Most brain malformations and maldevelopments comprise normal brain tissue arranged abnormally and are best detected with T1 weighted images. Foreign tissue including tumors and gliotic tissue is usually best detected with T2 weighted images[2]. Contrast agent gadolinium is rarely helpful and is not indicated as a routine. However, it can be helpful in conditions associated with breakdown in the blood-brain barrier such as tumors and vascular malformations[2]Detection of mesial temporal sclerosis, the pathological substrate underlying mesial temporal lobe epilepsy is usually not detected with standard T1 and T2 weighted axial images. The important features are decreased volume (atrophy) and increased signal on T2 weighted and FLAIR (Fluid attenuated inversion recovery) sequences in the hippocampus and / or amygdala. Its detection requires thin heavily T1 weighted and T2 weighted coronal images taken orthogonal to the long axis of the temporal lobe[2]

INDICATIONS FOR NEUROIMAGING: [55,9,53]

1. Focal onset of seizure at any age either in history or EEG
2. Generalized seizure in first year of life.
3. Focal or neurological deficits in examination.
4. If there is difficulty in controlling seizures with first line antiepileptic drugs.
5. Loss of seizure control with the current anti epileptic drug.
6. Change is seizure pattern, which may suggest some underlying lesion.
7. Children less than 2 years of age excluding simple febrile seizures.
8. New onset seizure / epilepsy presenting as a status epilepticus may need urgent neuroimaging.

MRI is considered as the imaging modality of choice than the CT because of superior anatomic resolution. CT has some advantages with identifying blood and calcifications. But MRI identifies more abnormalities than CT like cortical dysplasias, AV malformations, small tumors, however CT is more widely available than MRI and is less expensive and it does not require any sedation in young children due to very less timing.

OTHER NEUROIMAGING MODALITIES AVAILABLE ARE:[9,53]

1. **FUNCTIONAL MAGNETIC RESONANCE IMAGING (FMRI):**

   It can detect the focal changes in brain when certain area is activated, it can also detect the blood flow and oxygenation level

2. **POSITRON EMISSION TOMOGRAPHY (PET):**

   These scans can be performed during the ictal state to detect the focal areas of decreased metabolism; sensitivity is increased when it is performed soon after a seizure

3. **SPECT Scan:**

   Single photon emission computed tomographycan be useful when MRI is not very remarkable and it can yield the epileptic focus.

4. **MEG and MSI:**

   Magnetoencephalography is the recording of the magnetic fields generated by the intraneuronal electrical activity.

Magnetic source imaging is combination of MEG with Magnetic resonance imaging (MRI) .These both can be useful in pre surgical localization of epilepsy.

TREATMENT:[1,2]

- The decision of starting an antiepileptic must be made when there is no acute reversible cause of seizures.
- After an initial unprovoked seizure the decision to start an AED is individual one, but the risk of seizure reoccurrence must be considered.
- The aim of treatment is complete seizure control without any significant adverse effects. Anticonvulsants are decided based upon the individual seizure types.
- Anticonvulsants must be started initially with the low dose and it can be gradually increased till the seizure is fully controlled. Based on child's daily activity dosage can be adjusted.
- If there is no seizure control after the first AED second drug can be started and the initial drug can be tapered and stopped. The time period depends based on the drug reaching the therapeutic level.

The efficacy and tolerability of the 4 major anticonvulsants commonly used are same.

- Phenytoin
- Phenobarbitone
- Valproate
- Carbamazepine

- Therapeutic drug levels must be monitored periodically because certain drugs can cause severe adverse effects.

  E.g.: valparin causing liver damage

- In younger children (<2yrs) valproate must be avoided, drug interactions must also been checked specifically with AED's.
- Drugs with similar mechanism of action should also be avoided, because potentially there will be poor seizure control.

Therapeutic spectrum of antiepileptic drugs	
Broad spectrum:	
All seizure types (both focal and generalized onset)	
• Clobazam • Felbamate • Lamotrigine • Levetiracetam • Perampanel • Rufinamide • Topiramate • Valproate • Zonisamide	
Narrow spectrum (focal):	
Focal seizures only (including focal evolving to bilateral convulsive seizures*)	
• Carbamazepine • Eslicarbazepine • Ezogabine • Gabapentin • Lacosamide • Oxcarbazepine • Phenobarbital • Phenytoin • Pregabalin • Primidone • Tiagabine • Vigabatrin	

TABLE 1: Showing spectrum of AED's [7,8]

DISCONTINUATION OF THERAPY[1]

If the child is seizure free for more than 2 years AED's can be slowly tapered and stopped over a period of 6 months. Immediate discontinuation may result in recurrence of seizures.

EEG must be taken to see whether there is any abnormal electrical activity before discontinuation of the drug.

If there is recurrence with in 2-3 months then AED's must be restarted.

RISK OF RECURRENCE:[7,50]

A neurologically normal child has a risk of 24% after an first seizure and it up to 45% over the next 14 years.

Increased risk is usually associated with

1 Previous neurological insult

2 Significant MRI findings

3 Abnormal EEG

ALGORITHM FOR MANAGEMENT OF STATUS EPILEPTICUS[34]

Establish ABCs: Establish IV access, blood for investigations if needed.

↓

IV Lorazepam 0.1 mg/kg or IV diazepam 0.2 mg/kg, if no intravenous access buccal/nasal/PR diazepam/midazolam can be used

↓

Repeat Lorazepam/ Diazepam once more SOS (5-10mins)

↓

Intravenous fosphenytoin 20 PE (phenytoin equivalent)/kg/phenytoin 20 mg/kg

↓

IV valproate 15 to 20mg /kg loading may be given or phenobarbitone can be considered. Also airway to assessed if needed endotracheal intubation.

↓

Midazolam infusion (loading dose of 0.2 mg/kg)

↓

Paralysis if still seizure is not in control with propofol or thipentone

↓

continuous EEG monitoring can be done to know the seizure control.

Box 4: Unique side effects and toxicities of antiepileptic medications

- Phenytoin
  - Gingival hyperplasia, hirsutism, rash, osteopenia
- Carbamazepine
  - Hyponatremia, blood dyscrasias, allele HLA-B*1502 is associated with toxic epidermal necrolysis and Stevens-Johnson syndrome
- Felbamate
  - Liver failure, aplastic anemia
- Lamotrigine
  - Serious rash, worse with concurrent valproate
- Levtiracetam
  - Behavior or personality change
- Oxcarbazepine
  - Similar to carbamazepine
- Phenobarbital
  - Hyperactivity, aggressiveness, and insomnia in children, lowering of IQ, osteopenia
- Primidone
  - Similar to phenobarbital, more acute early toxicity that is transient
- Topiramate
  - Cognitive impairment, weight loss, paresthesias, kidney stones, metabolic acidosis, oligohidrosis
- Valproic cid
  - Hepatic toxicity, weight gain, tremor, polycystic ovaries, thrombocytopenia, tremor, hair loss
- Vigabatrin
  - Irreversible visual field defects, intramyelinic edema
- Zonisamide
  - Similar to topiramate, also do not use in sulfa-allergic patients

TABLE 2: Describes the common side effects of AED's

THE KETOGENIC DIET:[57]

Ketogenic diet can be helpful in children with uncontrolled seizures or in children with multiple antiepileptics. It is generally high fat (90%) very low carbohydrates and protein. The mechanism of this diet is not very clear. The understood mechanism is when high amount of fat is burned more ketones are produced. This lead to ketosis and it helps in by altering the amino acids and it increases GABA and leads to prevention of over firing of the nerve cells.

Ketogenic diet is very complex and difficult to follow. If the diet is stopped are discontinued suddenly it can lead in to a seizure. There are various studies are undergoing for the modification of diet, as children are finding it difficult to tolerate. Children must be regularly monitored when they are on ketogenic diet.

ADVANTAGES :[57]

Studies have shown that 10 to 15 % of children are seizure after 1 year of ketogenic diet. Also few children have stepped down their anticonvulsants afte initiation of ketogenic diet.

Early adverse effects: [57]

1 Acidosis

2 Hypoglycemia

3 Dehydration

4 Lethargy

LATE ADVERSE EFFECTS: [57]

1. Hyperlipidemia.
2. Vitamin deficiencies.
3. Renal stones.
4. Growth failure.
5. Reduced bone density.

This ketogenic diet can be continued for 2 years, but children should be monitored carefully during this diet.

SENSITIVITY OF NEUROIMAGING: [9,53]

Most of the persons with new onset seizures will not have a structural abnormality in MRI. A study shows the yield was 14%.Among various studies reported it ranges from 1 to 57% abnormal. These are mainly due to different technologies used either CT or MRI. As an example, emergency department based studies include a larger number with acute symptomatic seizures that are more likely to have corresponding CT or MRI abnormalities.

SPECIFICITY OF MRI: [9,53]

In individuals with idiopathic epilepsy MRI abnormalities are reported as 24%. But most of them were not epileptogenic, potentially epileptogenic lesions were seen in 3 to 4 percent. Similarly, in an another study reported 15% of which 71 children had abnormal MRI findings. Less than half of these findings were potentially epileptogenic.

A study, which was conducted in children with non-epileptic seizures MRI abnormality, was reported as 10 to 38% in this some were epileptogenic.

SENSITIVITY OF EEG:[10]

Ictalepileptiform discharges are found in 20 to 55 % in patients with epilepsy on a routine first EEG.The percentage increased to more 80% on further EEG's.

In one study Ictalepileptiformdischarges were 43 % in the first hour of recording.

SPECIFICITY OF EEG:[10]

The prevalence of Ictalepiletiform discharges in healthy children is about 3.5 to 6.5 % and in hospitalized adult patients its about 2 to 2.6%. but in children Ictalepileptiform discharges are less specific, in a series of study only 40% of children had epileptic seizures with mid temporal spike .

Akhterrasool et al,Suhil A. choh et al,[3] conducted a study on ole of neuroimaging and EEG in first onset afebrile seizures in children, and they found out 9.8% had abnormal CT and these were seen in patients with an abnormal EEG. MRI abnormality was 20.4% in patients with an abnormal EEG (24.8%). Most of the children with generalized and partial seizures had EEG abnormalities. These were common in age group between 6-14 years in this study.

Ramesh Baheti et al, BD Gupta et al,[4] has done a study of CT and EEG findings in patient with generalized or partial seizures. In this study they found out that EEG was abnormal in 73% and 76% of children with partial and generalized seizures. Abnormal CT scan was found in 50% of children with partial seizures and 34.6% with generalized seizures. The abnormality of CT scan increased with abnormal EEG.

Narenrdrasaini et al, AnamikaBaghel et al,[58] conducted a study on neuroimaging abnormalities in children with first afebrile seizure. They found out 66.7% of cases had abnormal neuroimaging and there was significant relationship between abnormal neuroimaging and focal seizure, which is 83.8% of children had abnormality.

Sharma et al, Rivelloet al[13]conducted a study on role of emergent neuroimaging in children with new onset afebrile seizures and they found 8% of abnormality in neuroimaging in the study group. 26% had clinically significant abnormality in the high risk group as described in the study.

Misra S et al,[14] has done a study to find out the Neuroimaging finding in children with seizure disorders and comparison of CT and MRI brain. A total of 96 children aged 3 months to 12 years presenting with seizure disorder were included. The cranial CT scan wasabnormal in 70% of the cases of seizure disorder. The incidence of CT scan abnormality was higher in focal seizures as compared to generalized seizures (78% vs 65%).

Murthy JM et al,[15]has done a study on etiological spectrum of 558 children (< 16 years) with partial seizures, and he suggeststhat in India a child with partial seizures with no obvious cause has a high probability of having an abnormal CT. In these patients, a CT scan should be the initial structural imaging investigation.

Hussainjageer et al,[5]did a study of cranial computed tomography in partial motor seizures. In this study 150 children with partial seizures were included, CT

scan was abnormal in 102 (68%) of children. Majority of children (75) had Single ring enhancing lesion in the parietal lobe.

Tarannum et al, Lateef et al, Rebecca et al[45]**,** did a study on neurologic complaints in young children and to determine the CT findings, CT scan was performed for around 394 children and they found 40% abnormality (154 children). In this 32 findings were significant.

Obajii MO et al,Fatunde OJet al[44], done a study on computed tomography and childhood seizures, the CT scans were abnormal in 51.5% of the study subjects. A high incidence of abnormal scan was reported in children with partial seizures.

Andrew J et al, Philip S et al,[49] done a study on magnetic resonance imaging findings in children with first recognized seizure and they found that out of 281 MRI's performed single abnormality was seen in 31% and two or more abnormalities in 12%. The most common abnormality reported in this study was ventricular enlargement 51%.

Susan amirsalari et al, Amin saburi et al, They had done cross sectional study in MRI findings in epileptic children and its relation to clinical and demographic findings. In this study a total of 200 children where investigated in that 196(98%) children had abnormal EEG, abnormal MRI was seen in 57(28.5%) patients

Abnormal MRI findings had significant relation with abnormal EEG, age, positive family history for epilepsy.

MATERIALS AND METHODS

STUDY PLACE : Department Of Pediatrics

PSGIMS&R, Coimbatore

STUDY DESIGN :

Part Prospective and part Retrospective - observational study.

STUDY POPULATION :

This study included children of age group between 1year to 15 years who presented with afebrile seizures as both outpatient and inpatient basis.

STUDY PERIOD : Retrospective from Jan 2011

Prospective July 2014 to July 2015

SAMPLE SIZE :

N= 4 x p x q /d2

N= 4 X 37 X 63 / 64 is 145

With an expected prevalence of 37% and allowable error of 20% of the Prevalence,

Out of 175 children with afebrile seizures 47 children were excluded, since those children did not satisfy the criteria (data or reports not available)

Total of 128 children were included for the study.

INCLUSION CRITERIA:

Children between 1 year to 15 years of age with afebrile seizures who are developmentally normal were included in the study. This includes children who presented with first afebrile or subsequent unprovoked febrile seizures.

EXCLUSION CRITERIA:

- Febrile seizures
- Developmental delay
- Seizure following trauma
- Neurologically abnormal children like mental retardation, cerebral palsy have been excluded from this study.

METHODOLOGY

Children who presents with afebrile seizures, initial management was done to stabilize the child according to the treating physician.

Child was recruited after getting an informed and written consent from the parents.

History and other details in the proforma are filled up for all the children. Complete neurological examination was done for all the children who are included in this study.

EEG was taken and the neurologist who was blinded to this study interpreted the report.

CT or MRI was done as per discretion of the treating physician.

The treating physician also decided the choice of anticonvulsants.

The radiologist interpreted MRI and CT scan reports and the results were documented. Radiologist was also blinded to this study.

These children were followed up in our OPD for seizure control.

For the retrospective data permission was obtained from hospital authorities for review of case sheets.

Children who were previously admitted with afebrile seizures are taken up for the study.

Proforma was filled from the available data in the case records.

Only case sheets with complete EEG results were included.

MRI & CT reports were available from Hospital system.

The number of anticonvulsants used was also noted from the case sheets.

DETAILS OF THE PROCEDURE:

EEG:

- EEG was done using 24channel RMS machine.
- It tookabout 15 to 30 min to record the EEG in children.
- In younger children's only photic stimulation is done. Older children hyperventilation sleep recording was also done.
- The Neurologist's interprets EEG readings.
- None of the patient in study group had video EEG recording.

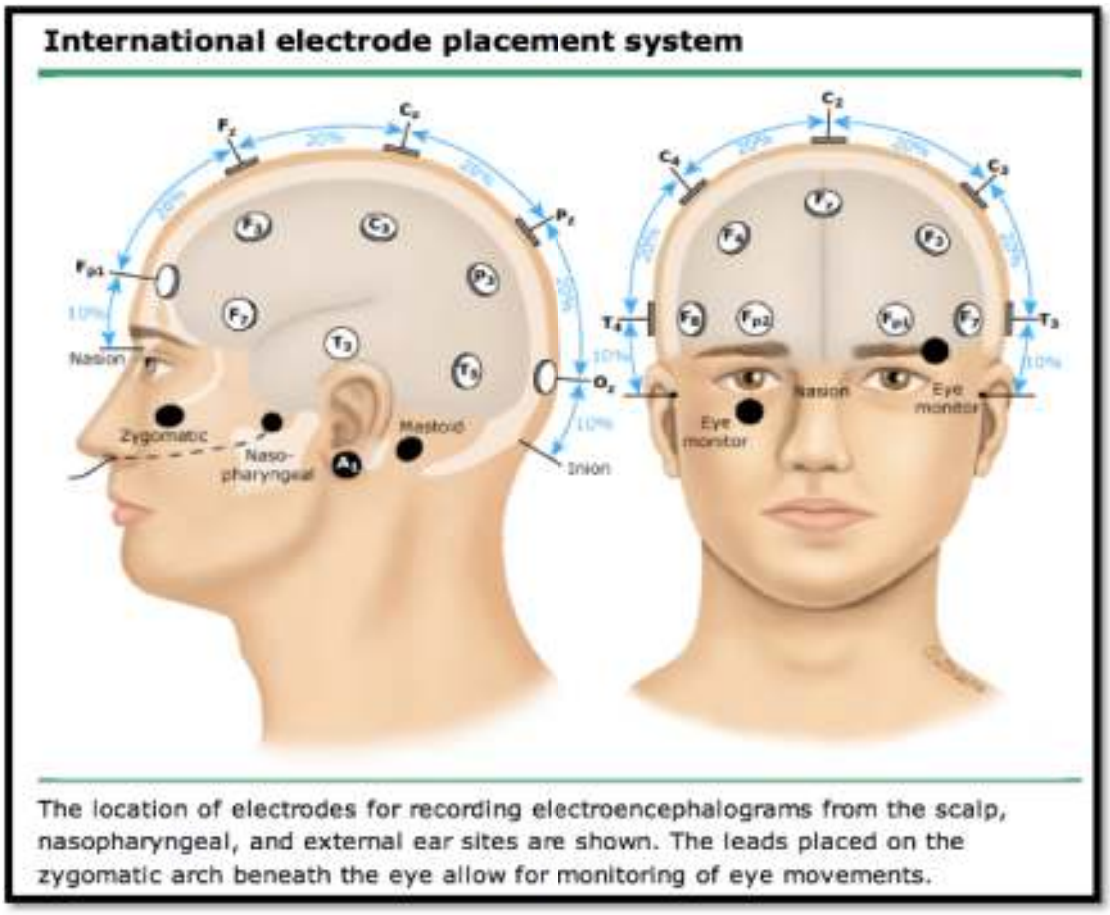

Fig 1: shows the location of electrodes placement [10]

NEUROIMAGING:

CT:

- This was done using SIEMENS 128 SLICE configuration.
- Contrast material was used as needed.
- The Radiologist interpreted CT findings.

MRI:

- SIEMENS 1.5 TESLA machine was used.
- Multiplanar, multisequence MR imaging of the brain was obtained.
- T1W1, T2W1 & FLAIR AXIAL , T1W1 SAGITAL, T2W1 CORONAL, DIFFUSION, ADC, SW AXIAL are the cut sections obtained.
- Short sedation either oral or intravenous is given for un cooperative and younger children's and their vitals are monitored throughout the procedure.

STUDY GROUP

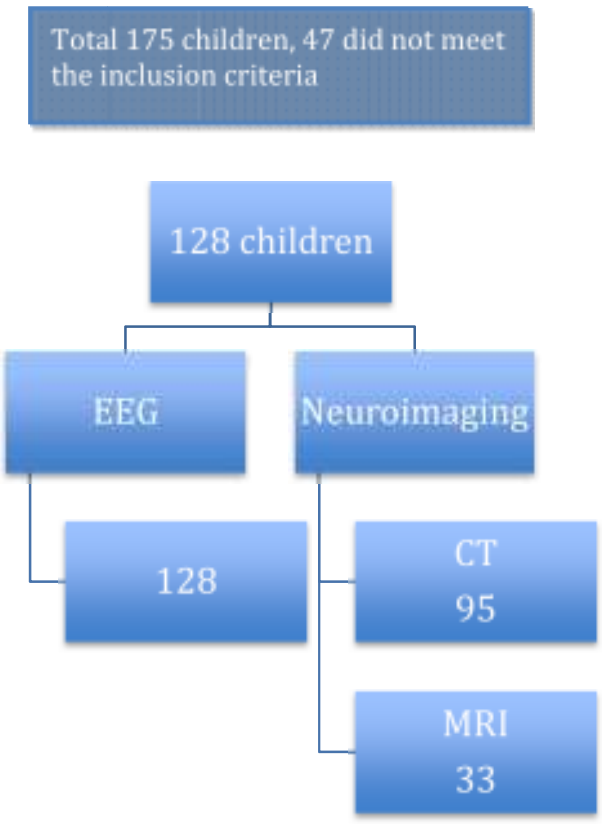

Fig 2: Showing study population

STATISTICAL ANALYSIS:

The data collected from the patients were formatted into Microsoft excel sheets to generate master charts, tables and graphs.

SPSS software was used to analyze data. Correlation of neuroimaging and EEG were assessed using chi-square Pearson co-efficient test.

RESULTS

AGE DISTRIBUTION:

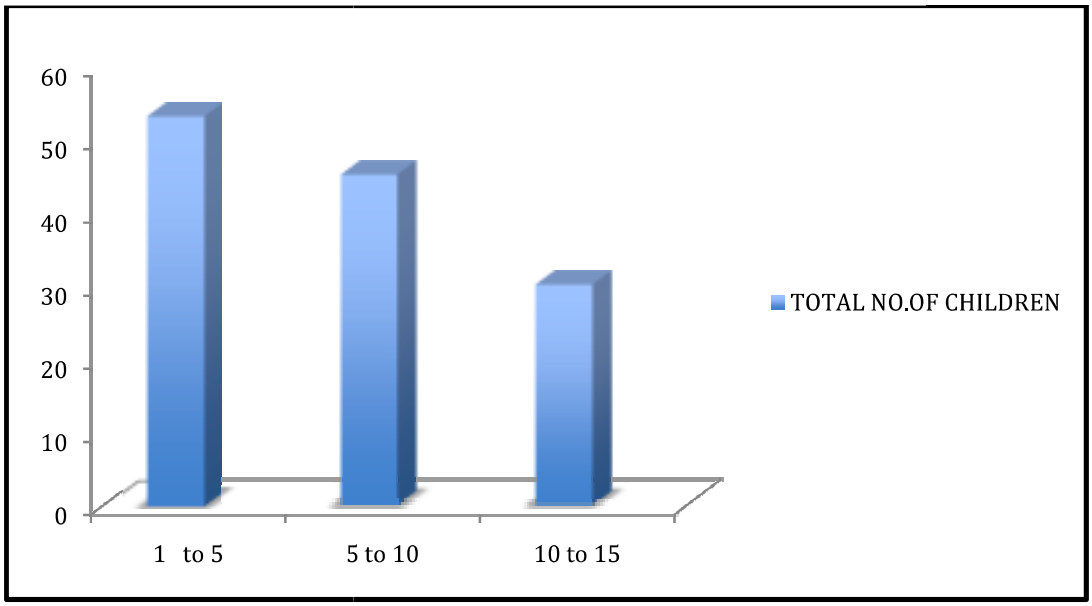

Chart 1: Age Distribution.

AGE IN YEARS	TOTAL NO.OF CHILDREN	%
1-5 years	53	41
5-10 years	45	35.1
10-15 years	30	23.4

Table 3: Age Distribution and percentage

Among our study population 53 children were in 1-5 years age group, 45 in 5-10 year age group and 30 children in 10-15 years age group.

SEX DISTRIBUTION

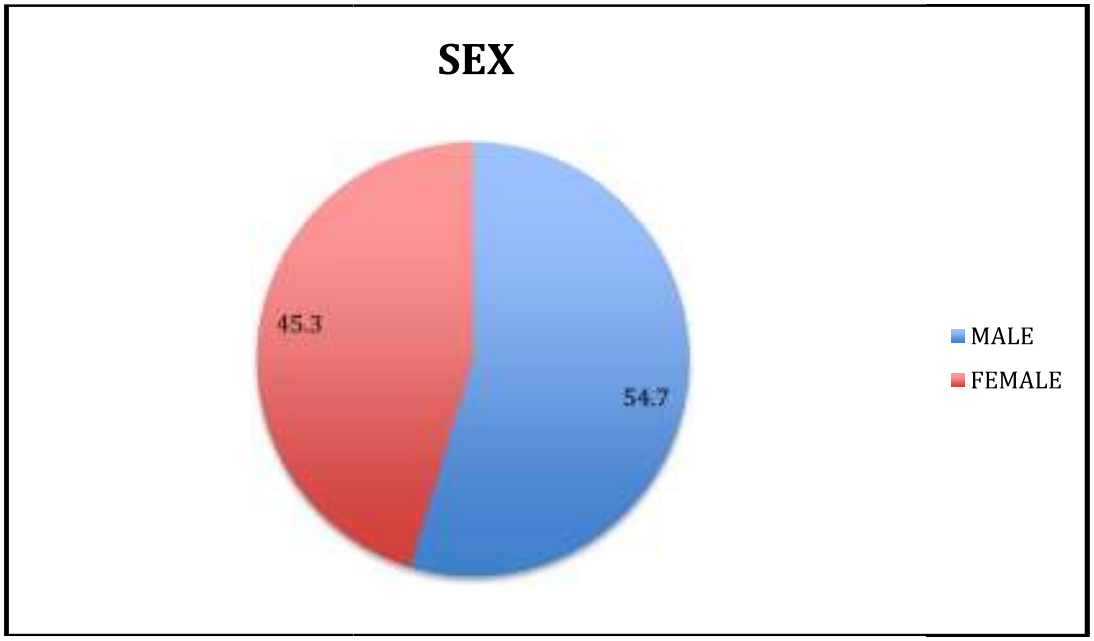

Chart 2: Sex Distribution

SEX	FREQUENCY	PERCENTAGE
MALE	70	54.7
FEMALE	58	45.3
TOTAL	128	100

Table 4: Sex distribution and percentage

This table describes male childrens were 54.7% (70/128) and female population is 45.3%(58/128) in our study.

FAMILY HISTORY OF SEIZURES

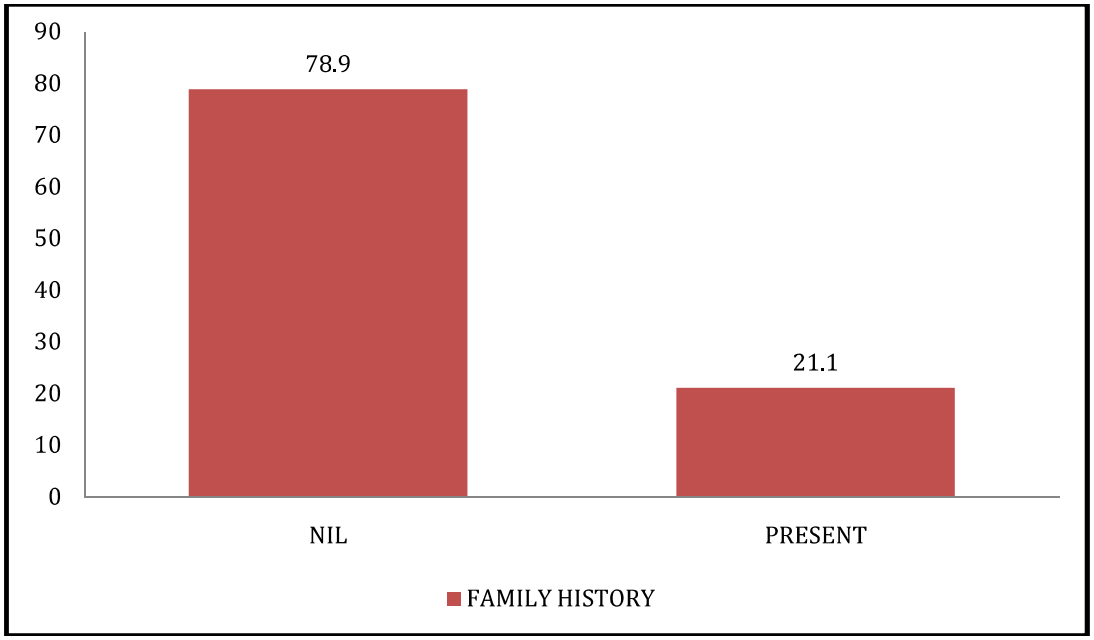

Chart 3: Family history frequency

FAMILY HISTORY	FREQUENCY	PERCENTAGE
NIL	101	78.9
PRESENT	27	21.1
TOTAL	128	100

Table 5: Family history frequency and percentage

This table shows family history was present in 21.1%(27/128) children in our study population.(p value <0.001).

TYPE OF SEIZURES

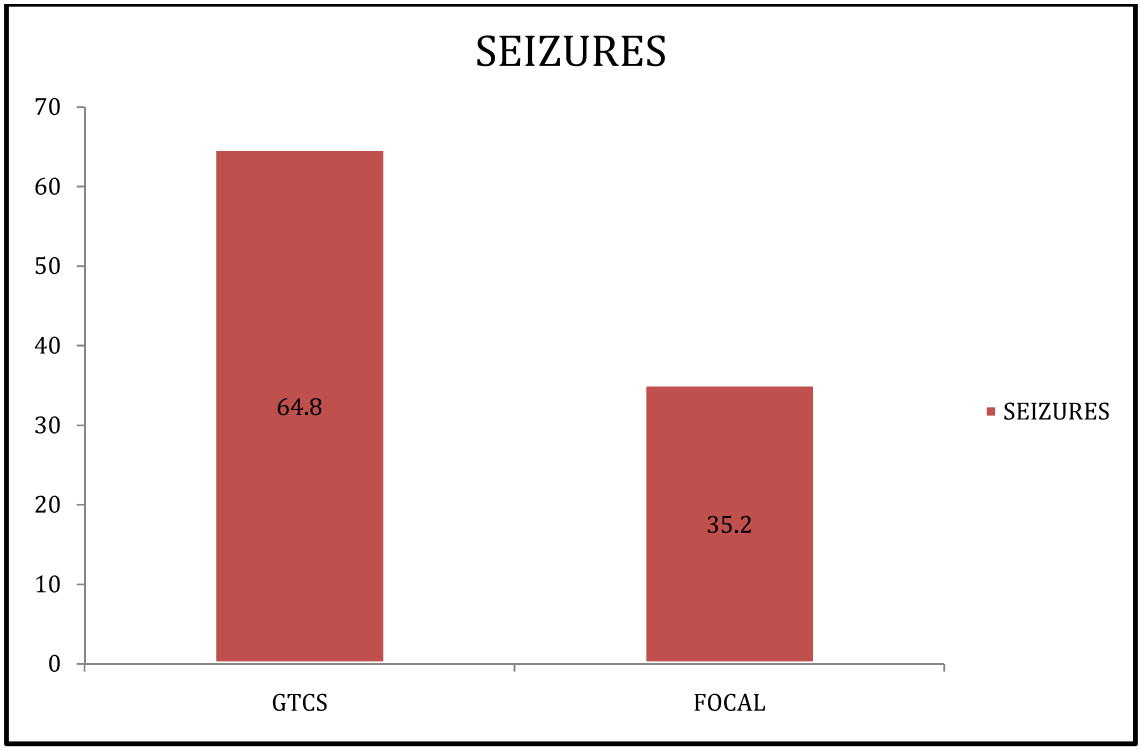

Chart 4: Type of seizures

Generalized seizure		**FOCAL seizure**	
NO	%	NO	%
83	64.8	45	35.2
INFERENCE: P VALUE <0.001			

Table 6: Type of seizures and percentage

This table shows generalized seizures are present in 64.8%(83/128) children and 35.2%(45/128) children

EEG FINDINGS

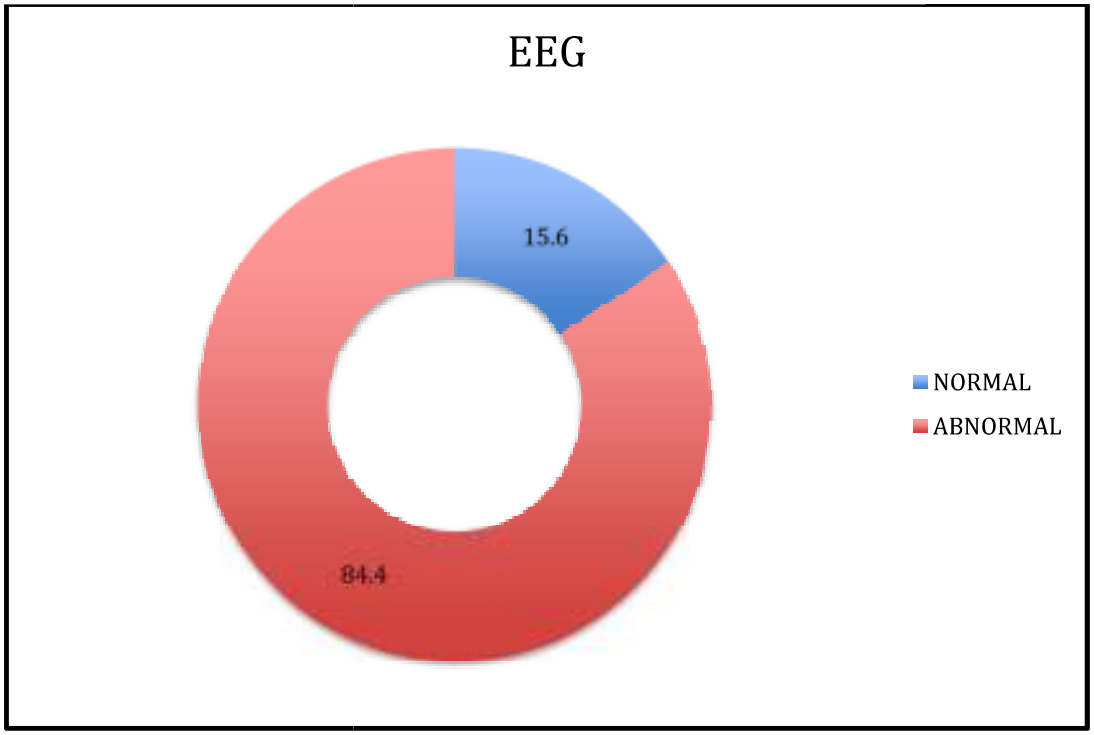

Chart 5: EEG abnormalities

NORMAL		ABNORMAL	
NO.	%	NO.	%
20	15.6	108	84.4
INFERENCE: P value < 0.001			

Table 7: EEG abnormalities and percentage

This table shows EEG was abnormal in 84.4%(108/128) children with afebrile seizures.

NEUROIMAGING

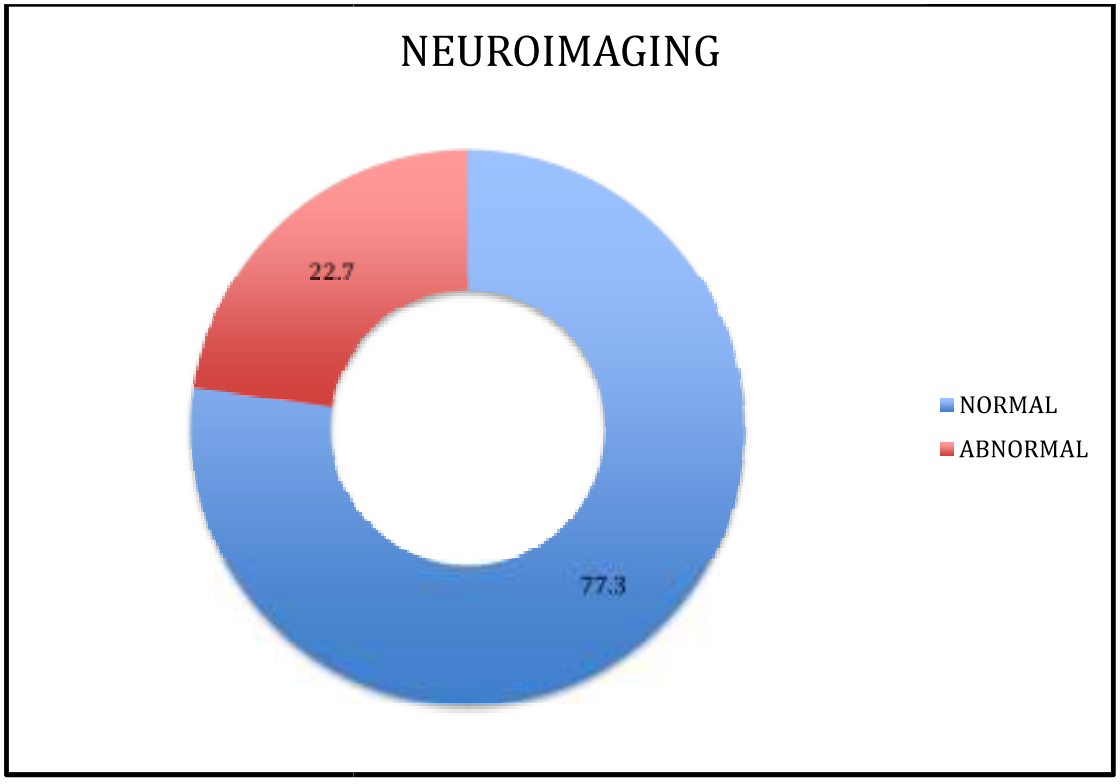

Chart 6: Neuroimaging abnormalities

NORMAL		ABNORMAL	
FREQUENCY	PERCENTAGE	FREQUENCY	PERCENTAGE
99	77.3	29	22.7
INFERENCE: P value <0.001			

Table 8: Neuroimaging abnormalities and percentage.

This table describes neuroimaging was abnormal in 22.7%(29/128) children in our study population.

NUMBER OF ANTIEPILEPTICS USED

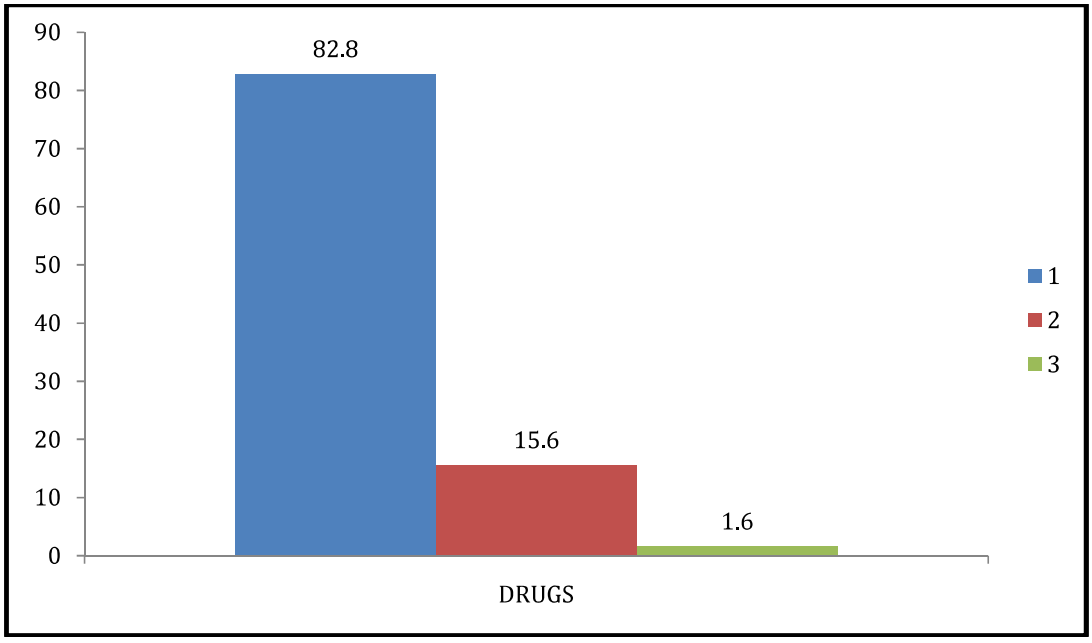

Chart 7: No. Of antiepileptics used

ONE		TWO		THREE	
NO.	%	NO.	%	NO.	%
106	82.8	20	15.6	2	1.6

Table 9: No. Of antiepileptics used

This table describes 82.8% with single antiepileptics and 15.6% with 2 antiepileptics and 1.6% with 3 antiepileptics in our study population.

SEIZURE TYPE IN AGE GROUPS

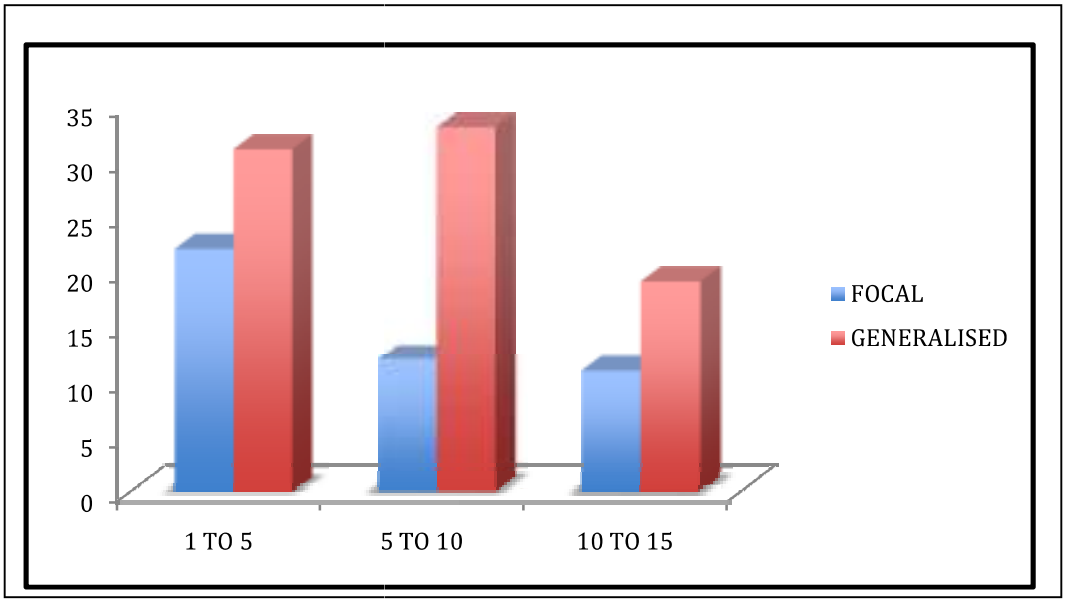

Chart 8: Type of seizure in age groups

AGE IN YEARS	FOCAL	GENERALIZED
1-5	22	31
5-10	12	33
10-15	11	19

Table 10: Type of seizures in diff age groups

In our study population 22 children are in 1-5 year group with focal seizures and 31 children with generalized seizures.

In 5-10 year age group 12 children with focal and 33 children with generalized seizures.

In 10-15 year age group 11 children had focal seizures and 19 children had generalized seizures.

GENDER DISTRIBUTION OF SEIZURES

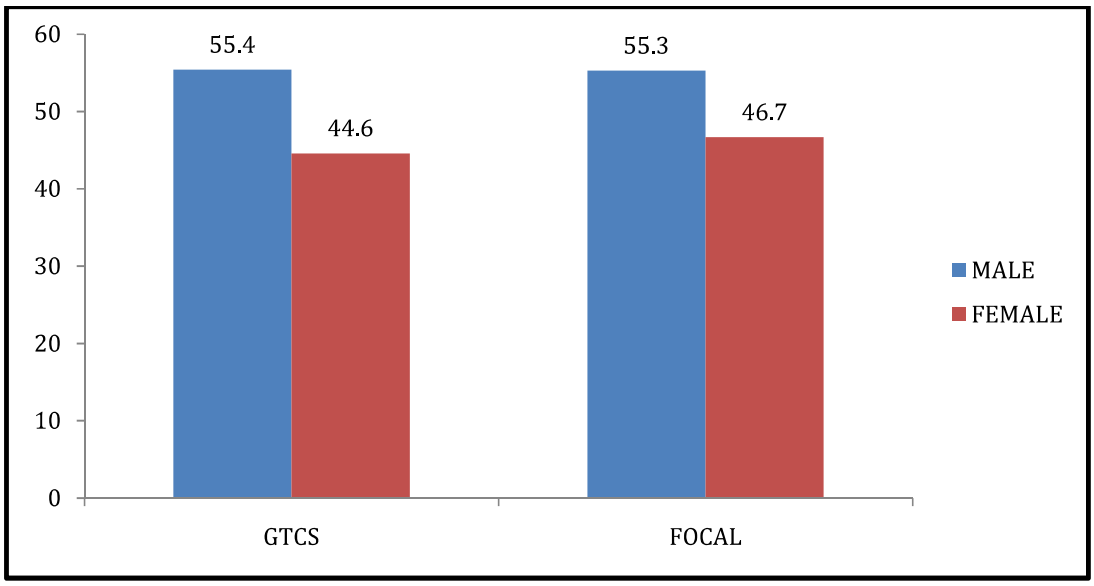

Chart 9: This bar chart describes the types of seizures present in the male and female children.

GENDER DISTRIBUTION OF SEIZURES:

SEX	GENERALIZED		FOCAL	
	NO.	%	NO.	%
MALE	46	55.4	24	53.3
FEMALE	37	44.6	21	46.7
TOTAL	83	100	45	100
INFERENCE:	P VALUE - 0.82			

Table 11: Type of seizure in male and female children.

This table shows generalized seizures are present in 55.4% of male children and 44.6% of female children and 53.3% of male children with focal seizures and 46.7% female children with focal seizures.

SEIZURE AND FAMILY HISTORY COMPARISON

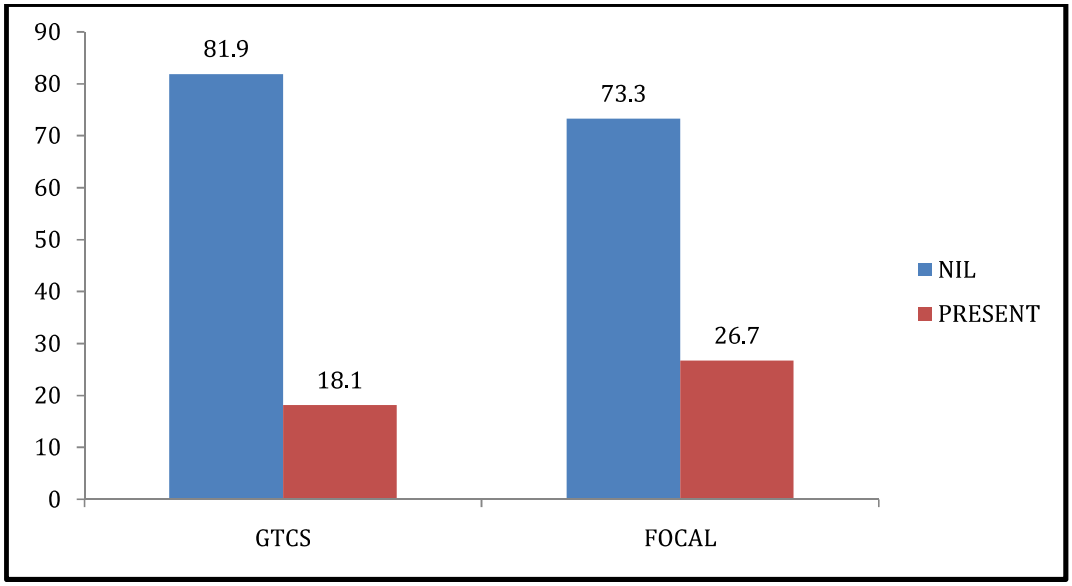

Chart 10: Type of seizure and percentage of family history

FAMILY HISTORY	GENERALIZED		FOCAL	
	NO.	%	NO.	%
NIL	68	81.9	33	73.3
PRESENT	15	18.1	12	26.7
TOTAL	83	100	45	100
INFERENCE	P= 0.26			

Table 12: Type of seizures with frequency and percentage of family history

This table describes positive family history is present in 18.1% with generalised seizures and 26.7% with focal seizures.

EEG & NEUROIMAGING ABNORMALITIES IN AGE GROUPS

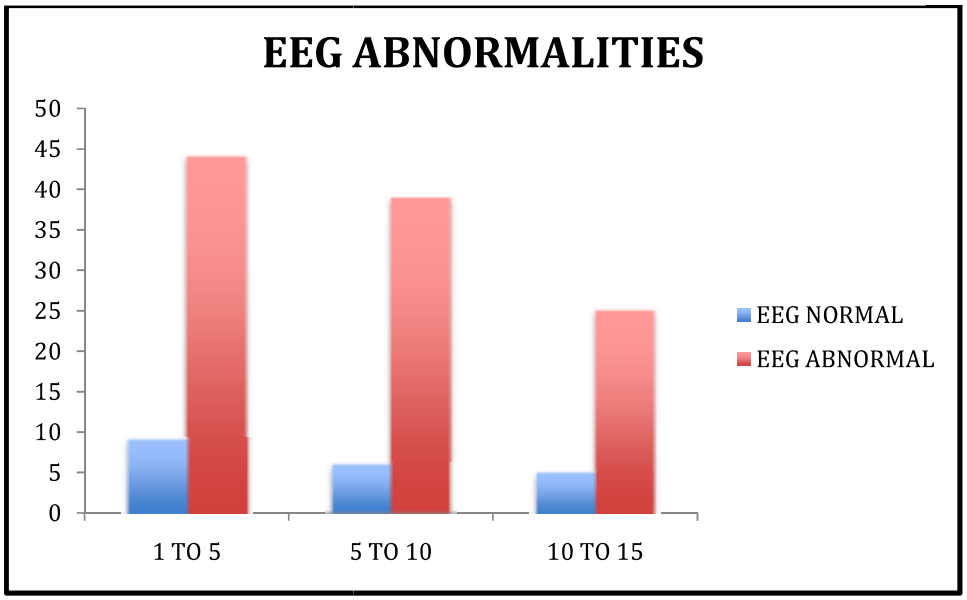

Chart 11: EEG abnormalities in age groups

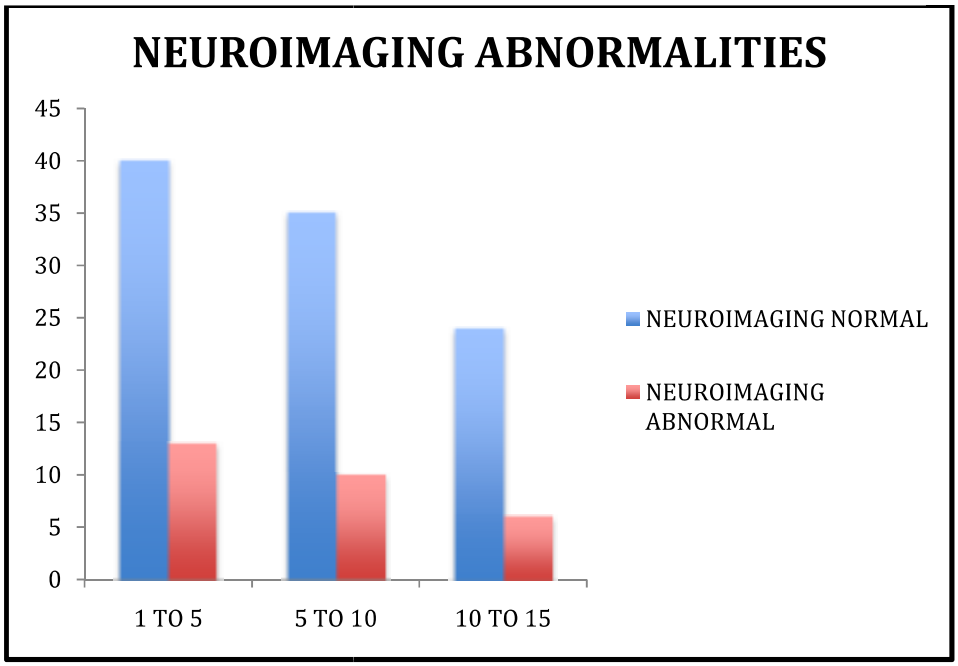

Chart 12: Neuroimaging abnormalities in age groups

EEG AND NEUROIMAGING ABNORMALITY IN AGE GROUPS

AGE IN YEARS	EEG		NEUROIMAGING	
	NORMAL	ABNORMAL	NORMAL	ABNORMAL
1-5	9	44	40	13
5-10	6	39	35	10
10-15	5	25	24	6
	20	108	99	29

Table 13: EEG and Neuroimaging abnormalities in Age groups

In 1-5 year old EEG is abnormal in 44 children and between 5-10 years 39 children had abnormality and 25 children in the age group of 10-15 years.

In 1-5 year old neuroimaging is abnormal in 13 children and 10 children in 5-10 year age group and 6 children in 10-15 years.

EEG ABNORMALITY IN CHILDREN WITH SEIZURES

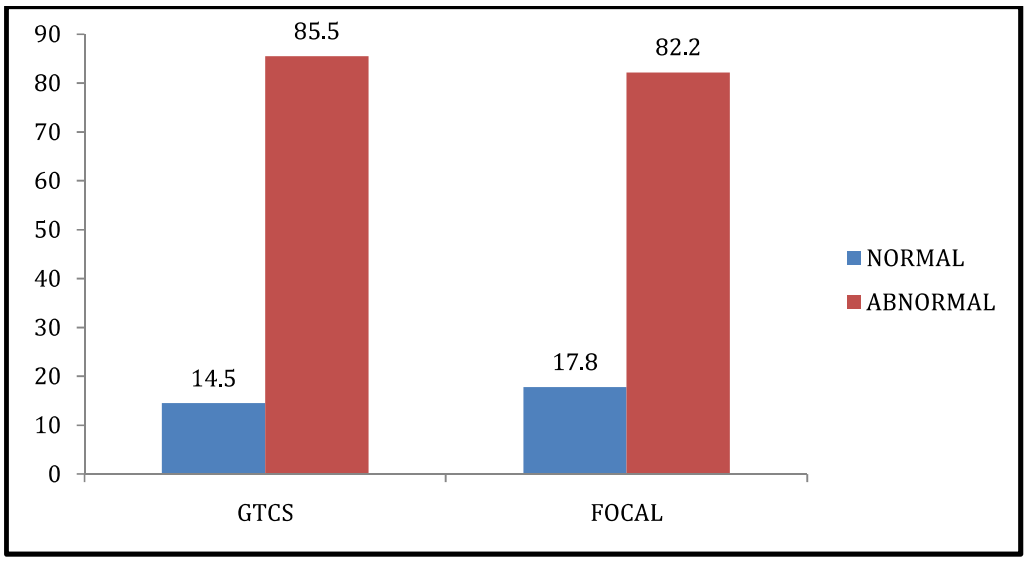

Chart 13: EEG abnormalities and type of seizures

EEG FINDINGS	GENERALISED		FOCAL		TOTAL
	NO.	%	NO.	%	
NORMAL	12	14.5	8	17.8	20
ABNORMAL	71	85.5	37	82.2	108
TOTAL	83	100	45	100	128
INFERENCE	P=0.62				

Table 14: EEG abnormalities with type of seizures.

This table describes abnormal EEG was present in 85.5% of generalized seizure and 82.2% with focal seizures in our study population.

The most common EEG abnormalities noted are as follows.

TYPE	TOTAL NO.	%
SHARP WAVES	17	13.2%
SHARP SPIKES	18	14.02%
BURST SPIKES	15	11.7%
B/L GENERALIZED EPILEPTIFORM ACTIVITY	56	43.7%
SLOW WAVES	2	1.56%
NORMAL	20	15.6%

Table 15: Type of EEG abnormality.

In our study group the most common EEG abnormalities noted are 43.7% of B/L generalized epileptiform activity, followed by sharp spikes (14.02%) and sharp waves (13.2%)

NEUROIMAGING ABNORMALITY IN CHILDREN WITH SEIZURES

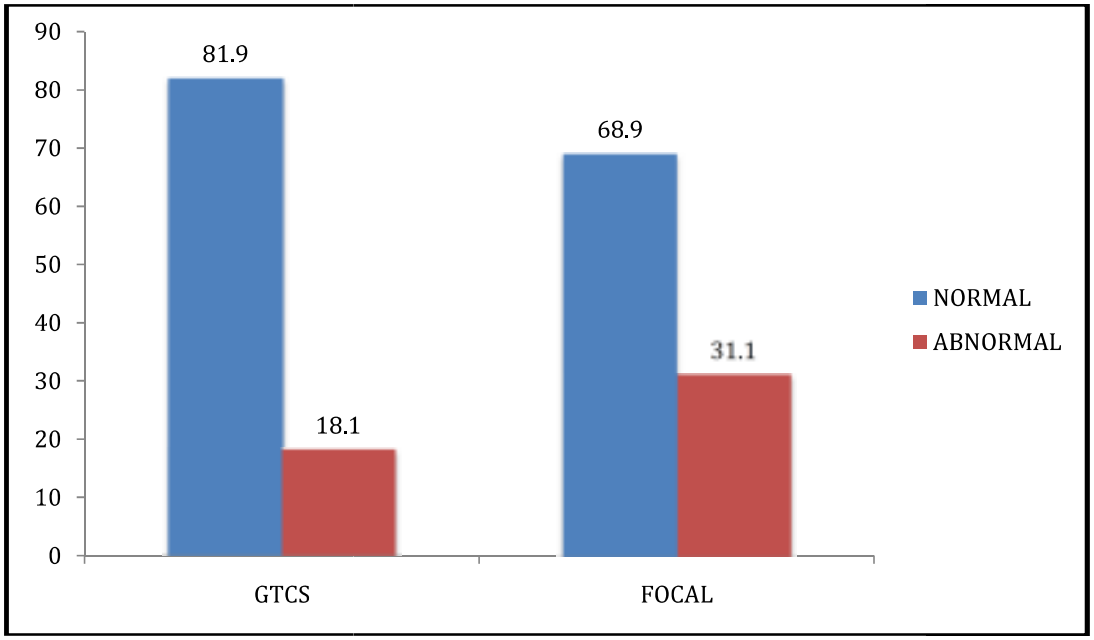

Chart 14: Neuroimaging abnormalities with type of seizures

NEUROIMAGING	GENERALIZED		FOCAL		TOTAL
	NO	%	NO	%	
NORMAL	68	81.9	31	68.9	99
ABNORMAL	15	18.1	14	31.1	29
TOTAL	83	100	45	100	128
INFERENCE	P=0.09				

Table 16: Neuroimaging abnormalities with type of seizures.

This table describes abnormal neuroimaging was seen in 18.1% of generalized seizures and 31.1% with focal seizures.

ANTIEPILEPTICS USAGE IN CHILDREN WITH SEIZURES

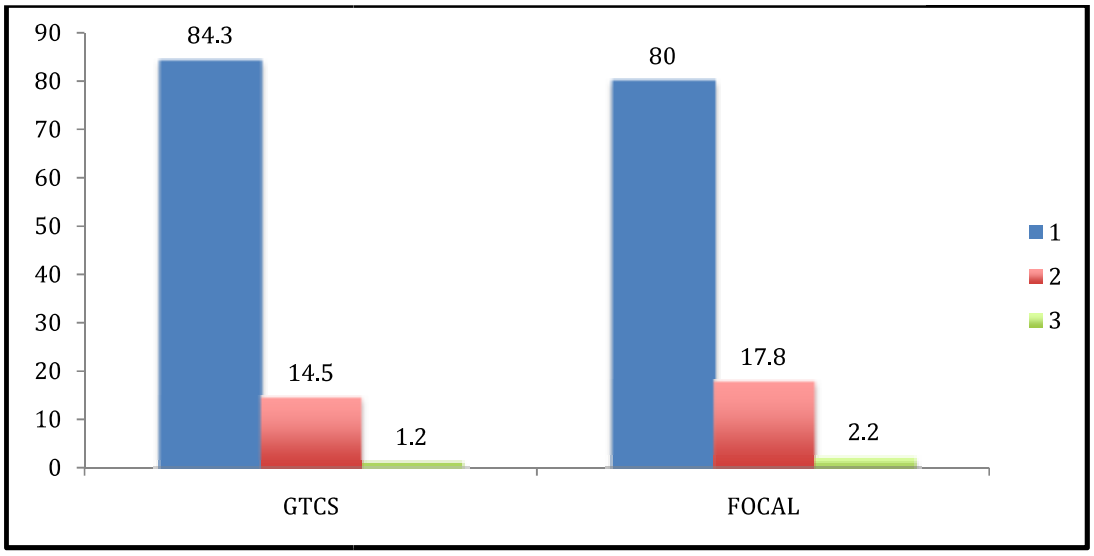

Chart 15: No. Of antiepileptics in type of seizures

DRUGS USED	GENERALIZED		FOCAL		TOTAL
	NO.	%	NO.	%	
1	70	84.3	36	80	106 82.8%
2	12	14.5	8	17.8	20 15.6%
3	1	1.2	1	2.2	2 1.6
TOTAL	83	100	45	100	128
INFERENCE	**P= 0.79**				

Table 17: No. and Percentage of antiepileptics in diff seizures.

This table describes 84.3% and 80% of children with generalised seizure and focal are on single antiepileptic. Where as 14.5% and 17.8% children are on 2 antiepilpetics.

COMMONLY USED ANTICONVULSANTS

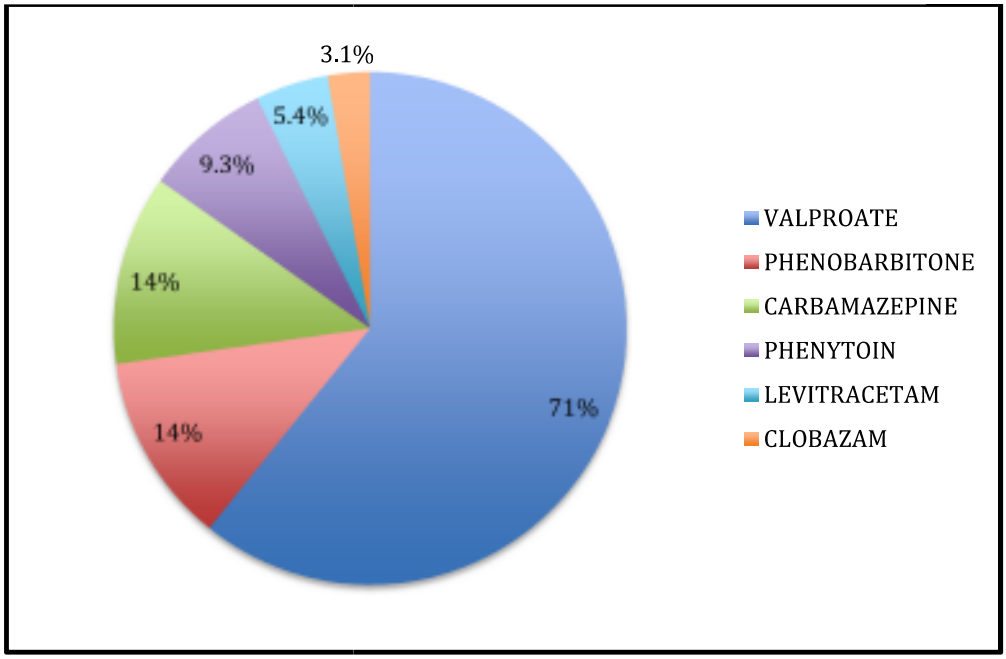

Chart 16: commonly used anticonvulsants

ANTIEPILEPTICS	%
VALPROATE	71%
PHENOBARBITONE	14%
CARBAMAZEPINE	14%
PHENYTOIN	9.3%
LEVITRACETAM	5.4%
CLOBAZAM	3.1%

Table 18: percentage of anticonvulsants used

In our study population the most commonly used antiepileptics are valproate (71%), followed by phenobarbitone, carbamazepine and phenytoin.

EEG AND NEUROIMAGING CORRELATION.

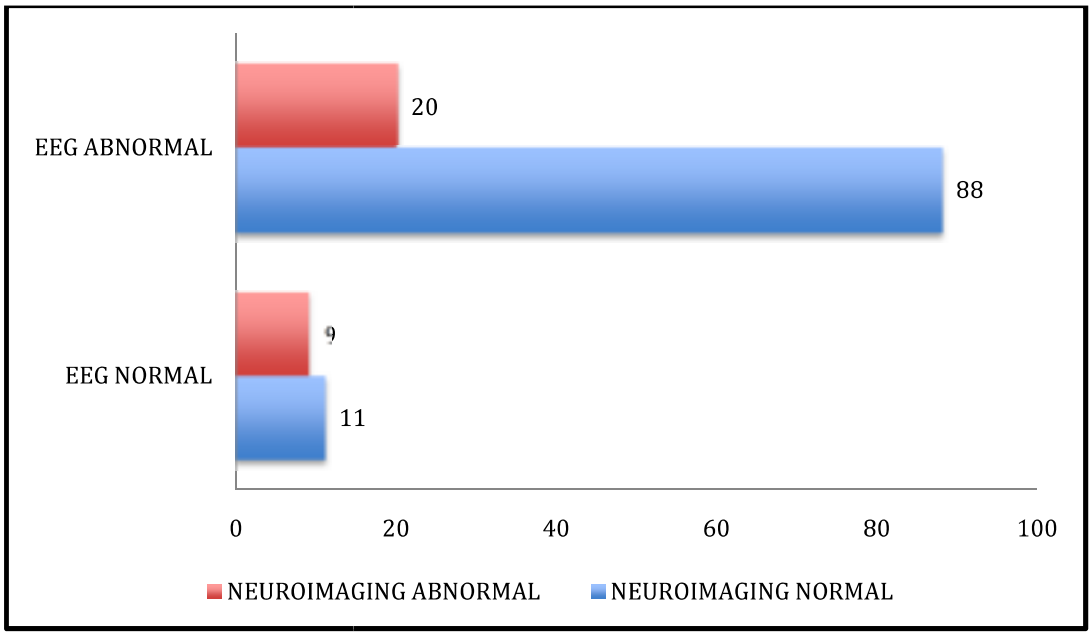

Chart 17: shows correlation between EEG and Neuroimaging in children with afebrile seizures.

EEG AND NEUROIMAGING CORRELATION

EEG/NEUROIMAGING		NEUROIMAGING		TOTAL
		NORMAL	ABNORMAL	
EEG	NORMAL	11	9	20
	ABNORMAL	88	20	108
TOTAL		99	29	128
INFERENCE		**P=0.009 Kappa -0.1**		

Table 19: correlation between EEG and Neuroimaging.

This table describes children with normal EEG 45% of children has abnormal neuroimaging (9/20). 20children with abnormal EEG have abnormal neuroimaging that is 22.7%.Statisticaly significant with a P value of 0.009 (<0.005 sig).

COMPARISON OF ANTIEPILEPTICS WITH EEG ABNORMALITY

NO. Of AED	NORMAL EEG	ABNORMAL EEG
1	17 (85%)	89 (82.5%)
2 OR MORE	3 (15%)	19 (17.5%)

Table 20: Comparison of AED's with EEG abnormality

COMPARISON OF ANTIEPILEPTICS WITH NEUROIMAGING ABNORMALITY

NO. OF AED	NORMAL NI	ABNORMAL NI
1	83 (82.5%)	24 (82.5%)
2 OR MORE	17 (17.5%)	5 (17.5%)

Table 21: comparison of AED's with neuroimaging abnormality

OBSERVATIONS:

- A prospective and retrospective study was conducted, which included 128 subjects, out of which 54.7%(70) were boys and 45.3%(58) were girls.
- The mean age noted is 6.1 with standard deviation of 4 and mean age of onset is 5.14.
- In our study population 41% of children belong to 1-5 year age group and 35.1% in 5-10 year age group and 23.4% belong to the 10-15 years age group.
- In the age wise category focal seizures were more common in 1-5 year age group children.
- In these children generalized seizures were noted in 83/128 that is 64.8% and focal seizures were present in 45/128, which is 35.2%.
- EEG abnormality was noted in 108/128 children which accounted for 84.4% and 20/128 had a normal EEG which is 15.6%.
- The most common EEG findings observed where b/l generalized epileptiform activity seen in 43.7% of children, sharp spikes seen in 14.02% of children and sharp waves in 13.2% of children, other findings like burst spikes (11.7%) and slow waves (1.56%) were less common.
- Neuroimaging was done in all of these children's and abnormalities were noted in 29/128, which is 22.7% of the total study population, and neuroimaging was normal in 99/128 that is 77.3%.
- Gliotic changes (27%) was the most common neuroimaging finding observed in our study.

- On the follow up of these children 82.8%(106/128), children were on single antiepileptic drug and 15.6%(20/128) of children on 2 antiepileptic's and only 1.6%(2/128) of children need 3 anticonvulsants.
- The common anticonvulsants used were valproate (71%), phenobarbitone and carbamazepine (14%) followed by phenytoin (9.3%), levitracetam(5.4%) and less commonly clabazam(3.1%).
- Family history of seizures were noted in 27/128 which is 21.1% and it was absent in 78.9%(101/128) of the study population.
- On comparing type of seizure and EEG 85.5% of EEG are abnormal in children with generalized seizure and 83.2% abnormality with focal seizures.
- When type of seizure and Neuroimaging are compared children with generalized seizures had 18.1%(15/83) had abnormality and 31.1%(14/45) with focal seizures had abnormality.
- 45%(9/20) of children with normal EEG had abnormal neuroimaging findings and 81%(88/108) of children with abnormal EEG had normal neuroimaging.
- Family history was positive in 18.1%(15/83) of children with generalized seizure and 26.7%(12/45) of children with focal seizures.
- On comparing number of antiepileptics with type of seizures, used in the study population, single antiepileptic is 84.3%(70/83) in generalized and 80%(36/40) of focal seizures.

- Children who required two anticonvulsants constituted4.5%(12/83) in generalized seizure and 17.8%(8/45) in focal seizures.1.2%(1/83) of children with generalized seizure had 3 antiepileptics and 2.2% (1/45) of children with focal seizures was on 3 antiepileptics.
- Even with abnormal EEG children requiring 2 or more anticonvulsants are similar with children having normal EEG.
- Anticonvulsant requirement; same in children with both normal and abnormal neuroimaging.

DISCUSSION

This study aims at the correlation of Neuroimaging and EEG in developmentally normal child with afebrile seizures; we also looked at the type of seizures and number of antiepileptics in these children.

Out of 128 children studied 70 children were male that is 54.7% and females were 58 that is 45.3%. Male predominance was noted in our study population which is comparable to study conducted by ReddaTekla – Haimanotet al[17].

The mean age of our study population is 6.1 years, which was comparable with ZajacA et al[6] and Ramesh Baheti et al[4]. The mean age of onset of seizures in these children's were noted to be 5.14 years.

In our study population age group was divided in to three sub categories as children between 1-5 years of age, 5-10 years and 10-15 years of age. Children between 1-5 years of age was more in our study group with 41% of the study population, and children between 5-10 years with 35.1%. comparing this with study done by AkhtherRasool et al, Suhil.A et al[3] where they observed children more than 6 years constituted in their study group with 63.8%.

Of the 128 children studied generalized seizures were noted in 64.8%(83) and 35.2%(45) presented with focal seizures. In our study population generalized seizures were more predominant than focal seizures. This result was comparable with a study done by Simi Misraet al[14] and ReddaTekla-Haimanot et al[17]. But AktherRasool et al and Suhil.Aet al[3] noted 44.3% of focal seizures and 47.7% of

generalized seizures. When compared with our study it showed equal predominance of both focal and generalized seizures.

Family history is present in 21.1% of children with afebrile seizures, when compared between seizures, generalized seizure had 18.1% that is 15 children had family history of seizures and 26.7% that is 12 children had positive family history in focal seizures. 21.1% of family history is comparable with ReddaTekla-Haimanotet al[17]where they showed 22%.

In our study population 108/128 children had abnormal EEG that is 84.4% and it was normal in 20/128 children with afebrile seizures that is around 15.6%. When comparing the abnormal EEG with seizures it is observed that child with generalized seizure has 85.5% abnormality that is 71/83 children. For focal seizures EEG abnormality was seen in 82.2% that is 37/45 children. When comparing our results with Ramesh Bahetiet al[4] where he observed 76.9% with generalized and 73% with focal seizures. This result is comparable with our study. KurupathRadhakrishanaet al[18] also noted around 74% EEG abnormality in children with generalized seizures in his study. But AkhtherRasool et al and Suhil.Aet al[3] noted 56.2% of EEG abnormality in their study. Most of the studies coincide with our study result. ShlomoShinnaret al[11] noted only 42% EEG abnormality in his study. Zajak et al[6]noted EEG abnormalities are more in children with focal seizures, but our study shows there is equal abnormalities noted in both focal and generalized seizures.

The most commonly observed EEG abnormality in our study population was B/L Generalized Epileptiform activity (43.7%), Sharp spikes (14.02%), Sharp waves (13.2%), followed by burst spikes (11.7%) less common EEG finding was slow waves which was seen only in 1.56% of our study population. When comparing this with study done by AkhtherRasool et al, Suhil. A et al[3] where they observed sharp waves in 19.8% of children and spike waves in 16.3% of children this is almost similar to our study.

Neuroimaging abnormality was noted in 29/128 children in our study population, which contributes to 22.7%. And 99/128 children had a normal neuroimaging. When comparing seizure and neuroimaging in our study 15/83 children with generalized seizure had neuroimaging abnormality, which is 18.1% and 14/45 children with focal seizure, had abnormality, which is 31.1%.

When comparing this results with other study AktherRasool et al and Suhil.A et al[3]noted 15.1% of children with focal seizure had neuroimaging abnormality, but Ramesh Baheti et al[4] noted 50% neuroimaging abnormality in children with focal seizure and 34.6% of neuroimaging abnormality in children with generalized seizures. Where as Obajiimiiet al[44] noted a total 51.5% neuroimaging abnormality and 74.4% abnormality for the children with focal seizures. Our study correlates with Shinnaret al[11] where he observed neuroimaging abnormality similar to our study which is 21%, our study also correlates with Azita et al[56] study where neuroimaging abnormality was 20%. And Taranummet al[45] observed 40 % abnormality in the studied population. The reason behind varying abnormalities of our study with the few studies observed are because our

study mostly included CT as the main modality of the neuroimaging, more abnormalities with focal seizure could be expected only with MRI. But our study correlates with the studies conducted with computed tomography.

Commonly observed neuroimaging abnormality in our study population is gliosis which accounted for 27%, others findings like cortical dysplasia, immature myelination, calcifications, septo optic dysplasia, infarct in the cerebral artery, arachinoid cyst, epidermoid cyst, and porencephalic cyst, as our study population included both CT and MRI we could not classify the neuroimaging findings, compared with studies, where they noted ring enhancing lesions more common. We had 1 case of neurocystecercosis, which was 3.4% of our study group, none of the children in our study group required surgical intervention during the study period.

The main aim of our study was to correlate the EEG and Neuroimaging findings, in 128 children of our study population, EEG was abnormal in 108/128 children and it was normal in 20/128 children, neuroimaging was abnormal in 29/128 children in our study population when comparing both the results of EEG and Neuroimaging.

Children with abnormal neuroimaging 108/128(84.6%) 18.5% that is 20 children had an abnormal Neuroimaging. Which is comparable with study conducted by AktherRasool et al and Suhil. Aetal[3], which showed 20.4% abnormality. In our study Neuroimaging was normal in 88/108 children with an abnormal EEG. This was comparable with study done by Ramesh Bahetiet al[4] where 70% of neuroimaging was normal.

Children with normal EEG 20/108 that is 15.6% neuroimaging was abnormal in 9/20 that is 45%. This is comparable with study conducted by AktherRasool et al and Suhil. A et al[4] where abnormal neuroimaging with a normal EEG is around 30 %, this shows that even children with an normal EEG has more chance of getting an abnormal neuroimaging finding. The correlation between neuroimaging and EEG in our study was statistically significant with an P value of 0.009 (<0.05 significant). Even with a small population group the neuroimaging abnormality increases with normal EEG.

So Neuroimaging should be considered in child with both normal and abnormal EEG. But type of seizure can also predict the neuroimaging findings. Annie T Berg et al[12] observed children with focal seizure has more chance of getting abnormal neuroimaging findings. But in our study abnormal EEG was similar in both focal and generalized seizures with 85.5% and 82.2%, so neuroimaging must be considered in all children who presents with afebrile seizures, EEG also helps in classifying the seizures in these children. The abnormality in neuroimaging is less in focal seizures as compared to other studies is mainly due to the modality of the imaging since most of the neuroimaging in our study group was CT. So MRI can detect more abnormalities in children with afebrile seizures. MRI must be considered in children who present with afebrile seizures with no other possible cause because the yield of abnormality in MRI is more as CT and there will also be less exposure to radiation in children.

We followed up our study population for their seizure control with no. of antiepilpetics they are on and we observed that 106/128 children were on single antiepileptics that is 82.8% and 20/128 children was on 2 antiepileptics which is 15.6% and only 2/128 children required 3 antiepileptics which is 1.6% of the total study population. Comparing this with the type of seizures, 70/83 children with generalized seizure that is 84.3% were on single antiepileptics and 4.5%(12/83) with 2 anticonvulsant and 1.2% with 3 anticonvulsants. Comparing with the focal seizure the number is almost same which is 80%(36/45) for single anticonvulsant and 8/45 with 2 antiepileptics and 1/45 that 2.2% with 3 antiepileptics.

Our result with number of anticonvulsant is comparable with Patrick kwan et al[21] and Martin.J et al[21] as they observed that 47% with single anticonvulsant and 14% with 2 antiepileptics, but there is no study to compare with type of seizure and their control with anitepilpetics.

The most commonly used anticonvulsants in our study population was valproate (71%) which is followed by carbamazepine and phenobarbitone with 14 % each, then by phenytoin which was used in 9.3% of our study population. Less commonly used antiepileptics in our study population is levitracetam and clobazam.

The outcome of EEG and neuroimaging, whether normal or abnormal doesn't affect the requiremnts of anticonvulsants. Most of children our study are requiring only single anticonvulsants.

In children with normal EEG, 45% (9/20) had an abnormal neuroimaging which significantly tells that the chance of abnormal neuroimaging with a normal EEG is high and neuroimaging must be considered in those children who presents with afebrile seizures, also the chance of getting abnormal neuroimaging with both focal and generalized seizure are increasing as seen in our study population.

LIMITATION

This is a part retrospective study and therefore a uniform protocol for imaging (like MRI) was not followed, uniform protocol for imaging will probably result in higher identification of abnormalities.

CONCLUSIONS

1. Among 128 children in the study 108(84.4%) had an abnormal EEG while, 29 (22.7%) out of 128 children had abnormal Neuroimaging.

2. The most common EEG abnormality was bilateral generalized epileptiform activity, which was seen in 43.7% of children, and the common neuroimaging abnormality was Gliotic changes, which was seen in 27% of children.

3. The Incidence of getting abnormal neuroimaging is similar in both focal and generalized seizure.

4. For seizure control 82.5% of children with abnormal EEG required only one AED.

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ABBREVATIONS

MRI	:	MAGNETIC RESONANCE IMAGING
EEG	:	ELECTROENCEPHALOGRAM
CT	:	COMPUTED TOMOGRAPHY
FMRI	:	FUNCTIONAL MAGNETIC RESONANCE IMAGING
AED	:	ANTIEPILEPTIC DRUGS
V EEG	:	VIDEO ELECTROENCEPHALOGRAM
PET	:	POSITRON EMSSION TOMOGRAPHY
SPECT	:	SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY
MEG	:	MAGNETOENCEPHALOGRAPHY
MSI	:	MAGNETIC SOURCE IMAGING
AV	:	ARTERIO VENOUS
IV	:	INTRAVENOUS
IM	:	INTRA MUSCULAR
FMRI	:	FUNCTIONAL MAGNETIC RESONANCE IMAGING

KEY FOR MASTER CHART:

AN	:	ABNORMAL
N	:	NORMAL
GEN	:	GENERALLIZED SEIZURE
FOCAL	:	FOCAL SEIZURE
+	:	POSSITIVE FAMILY HISTORY
_	:	NEGATIVE FAMILY HISTORY

EP	:	B/L GENERALIZED EPILEPTIFORM ACTIVITY
SH	:	SHARP WAVES
SP	:	SHARP SPIKES
BS	:	BURST SPIKES
O	:	EEG NORMAL
SW	:	SLOW WAVES
V	:	VALPROATE
PH	:	PHENOBARBITONE
P	:	PHENYTOIN
C	:	CARBAMAZEPINE
L	:	LEVITRACETAM
F	:	CLOBAZEM.
NI	:	NEURO IMAGING

PROFORMA

Name : Age : Sex :

Address:- Date :
Ip/op no.:

HISTORY

SEIZURE:

Age of Onset:

Duration of seizure

No. of episodes:

SEIZURE PATTERN : Tonic / Tonic – Clonic/ Myoclonic / Absence/ Focal/

- Postictal : Confusion / Headache / Vomiting / Weakness / Cranial Nerves
- Fever / Headache / Vomiting / Blurring of Vision

Birth History: Neonatal insult:

Family History:

Developmental history:

Drug History:

Head circumference:

CNS examination:

SEIZURE CLASSIFICATION (ILAE):

FOCAL CONSCIOUSNESS NORMAL

IMAPIRED CONSCIOUSNESS

GENERALISED

OTHERS –

INVESTIGATION:

CT BRAIN PLAIN

CONTRAST

MRI:

EEG

MEDICATIONS:

SEIZURE CONTROL:

SOP 03-V 3.0 / ANX 10-V 3.0

Institutional Human Ethics Committee
PSG Institute of Medical Sciences and Research, Coimbatore

Parental Consent Form

Title of Study: Neuroimaging and EEG in Developmentally normal children with afebrile seizures.

Name of the Principal Investigator: S. VIGNESH

Department: PAEDIATRICS

Your child is invited to participate in this study.

My name is **S.VIGNESH** and I am a Post Graduate at PSG Institute of Medical Sciences and Research, Coimbatore. This study is done to determine the CT/MRI and EEG findings of your child and to correlate these findings.

I am asking for permission to include your child in this study because I expect to have **Minimum145** participants in the study.

If you allow your child to participate, I will collect medical details from your child's case record.

Any information that is obtained in connection with this study and that can be identified with your child will remain confidential and will be disclosed only with your permission. His or her responses will not be linked to his or her name or your name in any written or verbal report of this research project.

Your decision to allow your (son/daughter/child/infant/adolescent youth) to participate will not affect your or his or her present or future relationship

with PSGIMS&R or PSG Hospitals. If you have any questions about the study, please ask me. If you have any questions later, call me at 9047311011If you have any questions or concerns about your (son/daughter/child/infant/adolescent youth)'s participation in this study, call 9047311011.

You may keep a copy of this consent form.

You are making a decision about allowing your child to participate in this study. Your signature below indicates that you have read the information provided above and have decided to allow him or her to participate in the study. If you later decide that you wish to withdraw your permission for your child to participate in the study, you can contact me.

You may discontinue his or her participation at any time. *This will not affect in any way your future treatment in this hospital.*

Name of the child:

Signature of Parent(s) or Legal Guardian with Date

Signature of Investigator with Date

பெற்றோரின் ஒப்புதல் படிவம்

ஆய்வின் தலைப்பு :

காய்ச்சலற்ற வலிப்பு தன்மை கொண்ட சாதாரண வளர்ச்சி உடைய குழந்தைகளின் நரம்புப்படவியல் (CT/MRI ஸ்கேன்) மற்றும் EEG (மூளைக்கான சுருள் படம்)

ஆய்வு மேற்கொள்பவர் :– கு. விக்னேஷ்

எனது ஆய்வின் வழிகாட்டி :– டாக்டர். ஜோதிலட்சுமி

துறை :– குழந்தைகள் நலத்துறை

இந்த ஆய்வில் நான் காய்ச்சலற்ற வலிப்பினால் ஏற்ப்படும் நரம்புப்படவியல் மற்றும் EEG மாற்றங்கள் குறித்து ஆராய்ச்சி செய்கிறேன்
உங்கள் குழந்தையை இந்த ஆய்வில் ஈடுபடுத்துமாறு கேட்டுக்கொள்கிறோம்.

என் பெயர் கு. விக்னேஷ் நான் பி எஸ் ஜி மருத்துவமனையில் முதுநிலை மாணவராக பயில்கிறேன். இந்த ஆய்விற்கு குறைந்தது 145 குழந்தைகள் பங்கேற்க வேண்டுமென்பதால் உங்களுடைய குழந்தையை இந்த ஆய்வில் பங்கேற்க அனுமதிக்குமாறு கேட்டுக்கொள்ளப்படுகிறது. நீங்கள் அவ்வாறு அனுமதிப்பதினால், நான் உங்களுடைய குழந்தையின் மருத்துவ குறிப்புகளை குறிப்பேட்டிலிருந்து ஆய்விற்காக பயன்படுத்திக் கொள்ள முடியும். அவ்வாறு சேகரிக்கப்பட்ட குறிப்புகள் இரகசியமாக வைக்கப்படும். பிற்காலத்தில் உங்களுடைய விருப்பத்தின்பேரில் மட்டும்தான் பிறருக்கு தெரியப்படுத்தப்படும்.

நீங்கள் உங்கள் குழந்தையை இந்த ஆய்வில் பங்குபெற அனுமதிப்பதினால் உங்களுக்கும் பி எஸ் ஜி மருத்துவமனைக்கும் உள்ள இணைப்பு எந்த விதத்திலும் பாதிக்கப்படாது. உங்களுக்கு ஏதேனும் கேள்விகள் அல்லது சந்தேகங்கள் இந்த ஆய்வைப் பற்றி இருந்தால் 90473 11011 என்ற தொலைபேசி எண்ணிற்கு அழைக்கவும்.

நீங்கள் இந்த படிவத்தில் இடும் கையொப்பம் உங்கள் குழந்தையை இந்த ஆயிவில் பங்கேற்க அனுமதிக்கிறீர் மற்றும் நீங்கள் இந்த படிவத்தை முழுமையாக படித்து புரிந்து கொண்டீர்கள் என்பதை உறுதிபடுத்துகிறது. நீங்கள் எதிர்காலத்தில் இந்த ஆய்விலிருந்து விலகிக் கொள்ள நினைத்தால் என்னிடம் தெரிவியுங்கள். நீங்கள் எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம். இதனால் உங்களுக்கு அளிக்கப்படும் சிகிச்சை எந்த விதத்திலும் மாறுபடாது.

குழந்தையின் பெயர்: தேதி :

பெற்றோர் / காப்பாளர் கையொப்பம் : ஆய்வாளரின் கையொப்பம் :

SOP 03-V 3.0 / ANX 09-V 2.0

Institutional Human Ethics Committee
PSG Institute of Medical Sciences and Research, Coimbatore

Assent to be in a Research Study
For children between 7-18 years old

Why are we meeting with you?

We want to tell you about something we are doing called a research study. A research study is when doctors collect a lot of information to learn more about something related to health and disease.. Dr vignesh and some other doctors are doing a study to learn more about EEG AND NEUROIMAGING IN DEVELOPMENTALLY NORMAL CHILDREN WITH AFEBRILE SEIZURES After we tell you about it, we will ask if you'd like to be in this study or not.

Why are we doing this study?

We want to find out the correlation between EEG and NEUROIMAGING
So we are getting information from lots of boys and girls like you.
In the whole study, there will be about 145 children.

What will happen to you if you are in this study?

Only if you agree, two things will happen:

1. CT/MRI scan of your brain
2. EEG to see the activity of your brain

Will this study hurt?

No this wont hurt you

Will you get better if you are in this study?

No, this study won't make you feel better or get well. But the doctors might find out something that will help other children like you later.

Will everybody come to know about my condition? (Confidentiality)

We will not tell other people that you are in this research and we won't share information about you to anyone who does not work in the research study

Is this bad or dangerous for me? (Risks involved)

No there is no risks involved

Do I get anything for being in the research?

No

Will you tell me the results?

Yes we will tell u the results when ever you want to know. This results will also be published in a book for research purpose. But we will not reveal your name in it .

Do you have any questions?

You can ask questions any time. You can ask now. You can ask later. You can talk to me or you can talk to someone else.

Do you have to be in this study?

No, you don't. No one will be mad at you if you don't want to do this. If you don't want to be in this study, just tell us. Or if you do want to be in the study, tell us that. And, remember, you can say yes now and change your mind later. It's up to you. *This will not affect in any way your future treatment in this hospital.*

Who can I talk to or ask questions to?

List and give contact information for those people who the child can contact easily (a local person who can actually be contacted). Tell the child that they can also talk to anyone they want to about this (their own doctor, a family friend, a teacher).

If you don't want to be in this study, just tell us. If you want to be in this study, just tell us. This will not affect in any way your future treatment in this hospital.
The doctor will give you a copy of this form to keep.

SIGNATURE OF PERSON CONDUCTING ASSENT DISCUSSION
I have explained the study to _____ in language he/she can understand, and the child has agreed to be in the study.

_____ _____
Signature of Person Conducting Assent Discussion Date

_____
Name of Person Conducting Assent Discussion (*print*)

Part 2: Certificate of Assent

I have read this information (or had the information read to me) I have had my questions answered and know that I can ask questions later if I have them.

I agree to take part in the research.

OR

I do not wish to take part in the research and I have not signed the assent below._____ (initialed by child/minor)

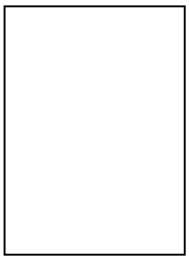
Only if child assents:
Print name of child _____
Signature of child: _____
Date:_____

If illiterate:

A literate witness must sign (if possible, this person should be selected by the participant, not be a parent, and should have no connection to the research team). Participants who are illiterate should include their thumb print as well.

I have witnessed the accurate reading of the assent form to the child, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

*Print name of witness (not a parent)*_____ *AND Thumb print of participant*
Signature of witness _____
Date _____



I have accurately read or witnessed the accurate reading of the assent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given assent freely.

Print name of researcher_____

மனித உரிமை கோட்பாடுகள் குழு
P.S.G. மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை
கோவை

இந்த ஆராய்ச்சி 7 முதல் 18 வயது திரும்பிய குழந்தைகளிடம் செய்யப்படுகிறது.

நாம் எதற்கு சந்திக்கிறோம்?

நாங்கள் ஆராய்ச்சி படிப்பைப் பற்றிக் கூற விரும்புகிறோம். ஆராய்ச்சி படிப்பு என்பது மருத்துவர்கள் ஒரு குறிப்பிட்ட மருத்துவ தகவல்களை சேகரித்து அதைப் பற்றி தெரிந்து கொள்வது.

நான் Dr.S.விக்னேஷ் காய்ச்சல் அற்ற வலிப்புனால் ஏற்படும் சாதாரண தன்மை கொண்ட குழந்தைகளின் நரம்பு படவியல் மற்றும் மூளைக்கான சுருள் படத்தை ஆராய்கிறேன்.

எதற்காக இந்தப் பரிசோதனை?

இதன் மூலம் C.T/ MRI SCAN மற்றும் E.E.G. பற்றி ஏற்படும் மாற்றங்களை தெரிந்து கொள்வதற்காக, இதற்காக நிறைய தகவல்களை ஆண் மற்றும் பெண் குழந்தைகளிடம் இருந்து பெறுகிறோம்.

இந்த ஆய்வில் மொத்தமாக 145 குழந்தைகள் உள்ளனர். இந்த பரிசோதனை மூலம் உங்களுடைய தொந்தரவுகள் சரிசெய்யப்படமாட்டாது.

ஆனால் இந்த பரிசோதனை மூலம் பிற குழந்தைகளுக்கு உபயோகமாக இருக்கும்.

உங்களின் பரிசோதனையின் முடிவுகள் வேறு யாருக்கும் செய்யப்பட மாட்டாது.

உங்களுக்கு மட்டுமே தெரியப்படுத்தப்படும் இந்த பரிசோதனையில் உங்களுக்கு சந்தேகம் இருந்தால் Dr.S.விக்னேஷ், 90473-11011

இந்த பரிசோதனையில் நீங்கள் பங்கு கொள்ள வேண்டும் என்ற கட்டாயம் இல்லை.

உங்களுக்கு விருப்பம் இல்லை என்றால் இதில் இருந்து எப்பொழுது வேண்டுமானாலும் விலகிக் கொள்ளலாம்.

இதனால் இந்த மருத்துவமனையின் எந்த சலுகைகளும் குறைக்கப்பட மாட்டாது.

Dr.S.விக்னேஷ் ஆகிய நான் இந்த சோதனையைப் பற்றி அனைத்து தகவல்களையும், குழந்தைகளின் பெற்றோர்களுக்கு தமிழில் கூறியுள்ளேன்.

குழந்தையின் பெற்றோர் அதை நன்கு புரிந்த பிறகு இதற்கு சம்மதம் தெரிவித்துள்ளனர்.

உங்களிடம் இந்த நோயைப் பற்றி சில கேள்விகள் கேட்கப்படும். புதிய பரிசோதனை எதுவும் செய்யப்படாது.

இந்தப் பரிசோதனை வழக்கமாக செய்யப்படும் பரிசோதனை மட்டுமே.

இந்த பரிசோதனையின் முடிவை மட்டுமே எனது ஆய்வுக்கு எடுத்துக் கொள்வேன்.

ஆய்வுக்குட்பட்டவரின் கையொப்பம். ஆய்வாளரின் கையொப்பம்.

தேதி:

பகுதி-2
சம்மத சான்று

நான் இந்தத் தகவலைப் படித்தேன் (அல்லது) தகவலை படிக்கக் கேட்டேன் எனது கேள்விகளுக்குப் பதில் அளிக்கப்பட்டது மற்றும் கேள்விகள் எதுவும் இருந்தால் அவைகளை வருங்காலத்தில் கேட்கலாம் என்பதும் எனக்குத் தெரியும்.

நான் இந்த ஆராய்ச்சியில் பங்கேற்க சம்மதிக்கிறேன்.

அல்லது

நான் இந்த ஆராய்ச்சியில் பங்கேற்க விருப்பம் இல்லை மற்றும் கீழே சம்மதம் என்று கையொப்பம் செய்யவில்லை. ----------.

(குழந்தை/மைனர்/இனிசியல் செய்யப்பட்டுள்ளது).

குழந்தை மட்டும் சம்மதம் தெரிவித்தால்

குழந்தையின் பெயர் :

குழந்தையின் கையெழுத்து :

தேதி :

படிப்பறிவு இல்லாதவராக இருந்தால் படிப்பறிவு உள்ள சாட்சி கையொப்பம் செய்ய வேண்டும் (முடிந்தால் இந்த நபர் கலந்து கொள்வபரால் தேர்ந்தெடுக்கப்பட வேண்டும். இந்த நபர் பெற்றோராக இருக்கக்கூடாது. மற்றும் அவருக்கு ஆராய்ச்சி குழுவிடம் எந்த தொடர்பும் இருக்கக்கூடாது).

நான் சம்மதப் படிவத்தை குழந்தையிடம் சரியாக படித்துக் காண்பித்ததைப் பார்த்தேன். மற்றும் அந்த நபருக்கு கேள்விகள் கேட்க வாய்ப்பு இருந்தது. அந்த நபர் தனது சம்மதத்தை முழு மனதுடன் கொடுத்தார் என்பதை நான் உறுதி கூறுகிறேன்.

சாட்சியின் பெயர் :

சாட்சியின் கையெழுத்து :

தேதி :

கலந்து கொள்பவரின் இடது
பெருவிரல் ரேகை

ஆராய்ச்சி செய்பவரின்

பெயர் : Dr.S.விக்னேஷ்

கைபேசி எண்.90473-11011.

NO.	OP NO	IP NO	AGE	SEX	SEIZURE	FAMILY	EEG	CT	MRI	NEURO	EEG	DRUG	NO OF	AGE OF
					TYPE	H/O				IMAGING			DRUGS	ONSET
1	O14073537	I14030924	6	F	GEN	-	AN	N		N	SH	V	1	4
2	O10073891	I14025919	9	M	GEN	+	AN	N		N	BS	V	1	5
3	O14066744	I14027395	10	F	GEN	-	N		N	N	0	C	1	10
4	O05046292	I14018958	11	M	GEN	+	AN	N		N	EP	L	1	3.5
5	O14046687	I14019745	7.5	M	GEN	-	AN	N		N	SP	V/P	2	7.5
6	O14029565	I14012344	2	F	GEN	-	AN	N		N	EP	V	1	1
7	O14031653	I14013313	6	M	GEN	-	N	N		N	0	V	1	6
8	O14025391	I14010397	1	F	GEN	-	AN	N		N	SP	PH	1	1
9	O14017076	I14006770	2	M	GEN	-	N	AN		AN	0	V	1	1.8
10	O09068605	I14005807	5	M	FOCAL	-	AN	N	N	N	EP	V/P	2	5
11	O14087274	I15007148	1	F	GEN	+	AN	N		N	EP	PH	1	1
12	O14078268	I15003507	11	M	GEN	-	AN	N		N	SH	P/L/T	3	10
13	O15003245	I15001493	4	M	FOCAL	-	AN	N	N	N	EP	PH	1	1
14	O14082070	I14034554	5	M	GEN	-	AN	N		N	BS	V	1	5
15	O09070250	I14036661	6	M	GEN	-	N	N		N	0	V	1	5
16	O12031342	I14034300	2	M	FOCAL	-	AN	N	AN	AN	EP	PH	1	2
17	O14076320	I14031855	13	F	GEN	-	N	N		N	0	V	1	12
18	O00042342	I15008873	14	F	GEN	-	AN	N		N	EP	V	1	14
19	O15010597	I15004745	9	F	FOCAL	+	AN		N	N	SP	V	1	9
20	O11018653	I15001227	5	F	GEN	-	AN		N	N	SH	V/P	2	3
21	O14028844	I14036071	1	M	GEN	-	AN	N		N	EP	V	1	1
22	O14087800	I14037100	5	M	GEN	-	AN		N	N	EP	V	1	1
23	O14038352	I14016167	14	M	FOCAL	-	N	N		N	0	V	1	14

24	O14074589	I15004157	3	M	FOCAL	-	AN	N	N	N	SP	V/L	2	2.5
25	O14082094	I14034598	10	F	FOCAL	-	AN	N		N	BS	C	1	10
26	O14079287	I14033211	3	M	GEN	-	AN	N		N	BS	P	1	3
27	O14069296	I14028557	10	M	GEN	-	AN		N	N	BS	V	1	10
28	O14069928	I14028854	9	F	FOCAL	-	N	N		N	0	V	1	9
29	O14025881	I14024021	4	F	FOCAL	-	AN		AN	AN	EP	V	1	3.5
30	O14015605	I14022966	8	F	GEN	-	AN	N		N	SH	V/P	2	7.5
31	O13039430	I14016854	1	F	FOCAL	-	AN	AN		AN	EP	V	1	1
32	O12091382	I14016893	7	M	FOCAL	-	N		AN	AN	0	V	1	1
33	O13031033	I14012998	10	M	FOCAL	-	AN		N	N	BS	V/C	2	10
34	O13020010	I13008544	12	M	GEN	-	AN	N		N	EP	C	1	11.5
35	O13090670	I13037922	13	F	GEN	-	AN	N		N	EP	V	1	13
36	O13089077	I13037148	7	F	GEN	-	AN	N		N	EP	V	1	7
37	O13030801	I13038100	1	M	GEN	-	N	N		N	0	PH	1	1
38	O13080329	I13033694	8	M	GEN	-	AN	N		N	SP	V	1	8
39	O13072097	I13030457	3	F	GEN	+	AN	N		N	EP	V	1	3
40	O13068449	I13028848	3	M	GEN	-	N	N		N	0	V	1	3
41	O04048667	I13023832	9	M	GEN	-	AN	AN		AN	SH	V	1	9
42	O13056069	I13023888	2	F	GEN	-	AN	N		N	EP	V	1	2
43	O13015583	I13015583	2	F	GEN	-	AN		AN	AN	EP	V	1	2
44	O10063683	I13002661	7	M	GEN	-	AN	N		N	EP	V	1	7
45	O14030698	I14012822	9	F	FOCAL	-	AN	N		N	SH	L/V	2	9
46	O14051960	I14020950	13	M	GEN	-	N		AN	AN	0	V	1	12
47	O14066784	I14027472	9	M	FOCAL	-	N		AN	AN	0	V/C/F	3	8
48	O13078653	I14056743	12	M	GEN	-	AN	N		N	EP	V	1	12
49	O15050277	I15022368	8	M	GEN	-	AN	N		N	SH	V	1	1.5
50	O15050821	I15022628	7	M	FOCAL	-	AN	N		N	EP	V	1	7

51	O12078622	I15024631	2	M	GEN	-	AN		AN	AN	SP	V	1	1.5
52	O13031717	I15022233	2	M	FOCAL	-	AN	N		N	EP	V	1	2
53	O11006104	I15023659	4	F	GEN	-	AN	AN		AN	BS	C/F	2	1
54	O15035072	I15021145	1	M	FOCAL	+	AN	N		N	EP	PH	1	1
55	O15038043	I15017302	4	M	GEN	-	AN	N		N	EP	V/L	2	1
56	O15038805	I15017684	10	M	GEN	-	AN	N		N	EP	V	1	10
57	O15033862	I15015435	8	M	GEN	-	AN	N		N	SP	V	1	8
58	O13058361	I15012683	9	F	GEN	-	AN	AN		AN	EP	P/C	2	2.5
59	O15025366	I15011553	15	F	FOCAL	+	AN	N		N	EP	P/C	2	15
60	O15026650	I15012506	1	M	GEN	+	AN	N		N	BS	PH	1	1
61	O14067563	I15011096	1	M	GEN	-	AN	N		N	BS	V/PH	2	1
62	O15026042	I15011935	9	M	GEN	-	AN		N	N	SP	V/P	2	8
63	O15049997	I15022226	9	F	GEN	-	AN	N		N	SH	V	1	7.5
64	O13088848	I15017426	1.5	F	GEN	-	AN	N		N	EP	V	1	1
65	O14018664	I15014179	4	F	GEN	+	AN	N		N	SH	V	1	3
66	O15024252	I15010995	1	F	GEN	-	AN	N		N	SP	V	1	1
67	O15026273	I15012064	5	M	FOCAL	+	AN	N		N	EP	V	1	5
68	O14074948	I15024997	1	M	GEN	-	N	AN		AN	0	V	1	1
69	O08023708	I15015304	7	M	GEN	-	AN		N	N	SP	V	1	1
70	O13068474	I15011281	1.5	M	GEN	-	AN	N		N	SP	V/P	2	1
71	O12012340	I12004801	7	F	GEN	+	AN	N		N	SP	L	1	7
72	O01039484	I12004138	10	M	GEN	+	AN	N		N	SP	V/F	2	7
73	O12016331	I12006572	6	F	GEN	-	AN	AN		AN	SH	V	1	4
74	O06016836	I12011301	10	F	GEN	-	AN	AN		AN	EP	V	1	10
75	O12038348	I12015322	13	M	FOCAL	-	AN	AN	AN	AN	BS	C	1	13
76	O12039314	I12015748	3	F	FOCAL	-	N	AN		AN	0	V	1	3
77	O09083241	I12016123	5	M	GEN	-	AN		N	N	EP	V	1	3

78	O12051003	I12020406	10	F	GEN	-	AN		N	N	SP	C	1	10
79	O12051943	I12020705	10	M	GEN	-	AN	N		N	SH	V	1	1
80	O12080716	I12032165	6	F	FOCAL	-	AN	AN		AN	BS	V	1	6
81	O12083060	I12033158	12	M	FOCAL	-	AN	N		N	SP	V	1	12
82	O12085173	I12034063	8	F	FOCAL	-	AN	AN		AN	EP	V	1	1.5
83	O10096677	I12035057	3	F	GEN	-	AN		N	N	EP	V	1	3
84	O12056733	I12024473	7	M	GEN	+	AN		N	N	BS	V	1	7
85	O08062737	I12027411	8	M	GEN	+	AN	N		N	EP	V	1	8
86	O12006725	I12008860	3	F	GEN	-	N	N		N	0	PH	1	3
87	O12035743	I12014172	3	M	FOCAL	+	AN		N	N	EP	PH	1	3
88	O09089172	I12034304	5	M	GEN	-	AN	N		N	EP	V	1	5
89	O08079810	I11035262	8	M	FOCAL	-	AN		AN	AN	EP	C	1	4
90	O11045430	I11035612	1	F	FOCAL	-	AN	N		N	SW	PH	1	1
91	O11087746	I11033157	13	F	FOCAL	-	AN	AN		AN	EP	C	1	8
92	O11083486	I11031539	14	M	FOCAL	+	AN	N		N	EP	L/F	2	14
93	O11072810	I11027456	4	F	FOCAL	+	AN	N		N	EP	V	1	4
94	O006015465	I11026450	6	M	GEN	-	AN	N		N	SH	C	1	6
95	O09064751	I11024148	2	F	FOCAL	-	AN	N		N	EP	PH	1	2
96	O07017010	I11021531	7	M	GEN	-	AN	N		N	EP	V	1	7
97	O11034415	I11021567	1	M	GEN	-	N	AN		AN	0	PH/P	2	1
98	O08011359	I11019124	5	F	GEN	-	AN		N	N	BS	C	2	2
99	O11053248	I11020477	6	F	GEN	+	N	N		N	0	V	1	6
100	O09055029	I11016529	4	F	FOCAL	+	AN		N	N	SH	V	1	4
101	O11044686	I11017175	14	F	GEN	-	AN	N		N	EP	C	1	14
102	O11031051	I11011960	11	F	GEN	-	AN	N		N	EP	V	1	10
103	O10103433	I11014635	3	M	GEN	+	AN	AN		AN	SH	PH	1	2
104	O11026597	I11010078	5	F	GEN	-	AN	N		N	SP	PH	1	5

105	O11019805	I11007384	1	F	FOCAL	+	AN	N		N	SH	V	1	1
106	O11008583	I11003112	7	F	GEN	+	AN	N		N	SH	V	1	7
107	O11011091	I11OO4103	11	M	FOCAL	-	AN	N		N	EP	P/V	2	4
108	O11009115	I11003321	10	F	GEN	+	AN	N		N	EP	V	1	10
109	O11005335	I11002124	2	F	FOCAL	-	N	N		N	0	PH	1	2
110	O11000591	I11000257	10	F	FOCAL	-	AN	N		N	BS	V	1	10
111	O10100048	I11033749	8	F	GEN	-	AN	AN		AN	EP	V	1	7
112	O10102679	I11034179	3	M	FOCAL	-	AN	N		N	BS	V	1	3
113	O11093806	I11035365	1	M	FOCAL	-	N	AN		AN	0	V/PH	2	1
114	O11088665	I11033475	4	M	GEN	-	AN	N		N	EP	V	1	4
115	O08077914	I11030378	5	M	GEN	-	AN	N	N	N	SP	V	1	5
116	O11074223	I11027901	2	F	FOCAL	+	AN	N		N	EP	V	1	2
117	O11032118	I11027797	1	M	GEN	-	AN	N		N	SH	V	1	1
118	O10095077	I11023417	2	F	GEN	-	AN	N		N	EP	V	1	2
119	O11060604	I11022909	4	M	GEN	-	AN		N	N	EP	V	1	1
120	O10032294	I11017344	1	M	FOCAL	-	AN		N	N	EP	PH	1	1
121	O11041618	I11015936	1	M	GEN	-	AN	AN		AN	EP	V	1	1
122	O09090576	I11012799	1.5	M	FOCAL	+	AN	N		N	SP	V	1	1.5
123	O13075098	I13031478	10	M	FOCAL	-	N		AN	AN	0	C	1	10
124	O13058361	I13024825	8	F	FOCAL	-	AN	AN		AN	EP	V	1	1.5
125	O02015207	I13021167	13	F	GEN	-	AN	AN		AN	SW	C	1	13
126	O10098713	I13004505	2	F	GEN	+	AN	N		N	EP	V	1	2
127	O13050123	I13021457	4	F	FOCAL	+	AN		N	N	EP	V	1	1.5
128	O13044637	I13018221	2	M	FOCAL	-	AN	N		N	EP	PH	1	2